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PRESIDENT'S ADDRESS

AMERICAN ASSOCIATION OF ORTHODONTISTS

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YOU have just listened to an address of welcome by our distinguished guest, Dr. Franklin Bliss Snyder, President of Northwestern University. As your President, I wish to repeat this welcome to the Thirty-Eighth Annual Meeting of the American Association of Orthodontists. We, your hosts in Chicago, sincerely hope that your attendance will be both pleasant and profitable. If this meeting is a success, no credit is particularly due me. It was my good fortune to obtain the services of committeemen who gave generously of their time and ability.

According to the Constitution and By-Laws of the American Association of Orthodontists, it is my duty to deliver a President's Address. It is customary for the President of any organization to be somewhat ponderous and philosophical as he views the world around him, so I will likewise comment on some of the current "Isms" that confront us at the moment.

There is an article in the *Reader's Digest* called "The Parable of the Isms" which goes as follows:

> Socialism: If you have two cows, you give one to your neighbor.
>
> Communism: If you have two cows, you give them to the government, and the government then gives you some milk.
>
> Fascism: If you have two cows, you keep the cows and give the milk to the government, then the government sells you some milk.
>
> New Dealism: If you have two cows, you shoot one and milk the other; then pour the milk down the drain.
>
> Nazism: If you have two cows, the government shoots you and keeps the cows.
>
> Capitalism: If you have two cows, you sell one and buy a bull.

Thirty-Eighth Annual Meeting, Chicago, Ill., May 13 to 16, 1940.

In this country, we are still operating under the capitalistic system, which implies that if we are to survive, we must make a profit. It is not so long ago that a professional man who talked about the business side of his profession was considered a heretic and was nearly ostracized from so-called good society. We are finally waking up to the fact that the business side of orthodontics has a definite place besides the dark recesses of a closet. I am sorry that the fullness of our program does not permit a presentation on this subject. Possibly a better place for such a discussion is in the smaller meetings of the different component societies. At any rate, I believe it might be a good idea to pull the old skeleton out and rattle its bones a bit. Too many of our men have labored sincerely and diligently for years only to find their declining years fraught with financial worries.

The practice of orthodontics is a profession, a method of making a livelihood and a way of life from which intangible rewards come to him who relieves human suffering and human disfigurement. This triad of profession, business, and way of life is inseparable. No man can do the professional job well if hanging over his head is the threat of economic want and privation. The business side of orthodontics should never be overstressed as something separate and apart. We should recognize that only those orthodontists who are economically successful can do the professional job with the skill and attention it deserves. The shadows of worry and want are not conducive to satisfactory professional attention.

The earning potentialities of professional men are among the highest in the population. Not only must professional men be taught how to apply their skill in fashions to make honest earnings, but they must also be taught, as early in their career as possible, the need of conservation of their earnings. Dental schools in general have been sadly remiss in preparing their graduates for the realities of actual practice. They have tossed young men into the practical world, and some of these men in turn have handled, during their practices, sizable sums of money, much of which was squandered in extravagant living and worthless investments. Why is it that professional men head the so-called "sucker list" of all fly-by-night investment concerns? Not for a minute would I belittle our scientific advancement, but I do feel that we owe it to the younger men to help them avoid the pitfalls of economics as well as helping them avoid the pitfalls of science.

The practice of orthodontics implies a relationship with the general practitioner of dentistry. No efforts should be spared to keep this relationship as cordial as possible. The method of reference on the part of the general practitioner, his statement to the patient, his method of explaining in general terms the often vexatious fee question, are important factors in the success of the proposed treatment.

Nor should the orthodontist lose sight of his responsibility in this relationship. He should make constant and strenuous efforts to keep patients returning to the general practitioner for examination and the treatment of early lesions. When the life, the personality, and appearance of a child are at stake, we cannot risk any misunderstandings among the parties involved—the patient and his parents, the general practitioner, and the orthodontist.

It might be well for all of us if general practitioners were more frequently invited to address our orthodontic meetings. Many general practitioners have shown an interest in learning more about orthodontics. Many of these men can afford neither the time nor the money to enroll for graduate or post-graduate courses. They turn then to short study club courses sponsored by dental societies, or, worse still, they cast themselves before the mercy of the commercial laboratories. It is the responsibility of orthodontic organizations to assure interested general practitioners methods of instruction that are scientifically sound. In communities where dental societies are undertaking study club courses in orthodontics, members of the American Association of Orthodontists should be the guiding spirits and the teachers. To do less is to leave the general practitioner a victim prostrate before unqualified and unscrupulous teachers and commercial laboratories.

It has been my good fortune and pleasure to attend four sectional meetings of the seven components of our Association. Without exception, these meetings were well attended, and the component societies showed themselves to be in a very healthy and live condition. I believe it to be a good policy for the President of the American Association of Orthodontists to visit as many of the components as possible during his term of office. To do this requires a great deal of traveling and absence from his office. I would recommend that the Chairman of the Budget Committee be empowered to set up in the Annual Budget an amount of money to cover traveling expenses of the president. If the president is willing to devote his time to making these trips, he certainly should be reimbursed for his expenses.

I would also recommend that as long as each sectional society is holding an annual meeting, and the parent body is also to continue to hold an annual meeting, the sectional meetings should all be held in the fall or early winter in order not to conflict with the annual meeting of the American Association. I am sure that our attendance at this meeting has been curtailed because of the close proximity of component meeting dates. We are holding our meeting later this year than usual because we hoped for a better break in the weather. We are extremely sorry that our meeting dates have conflicted with those of some of our state societies. It is inevitable that there will be some conflict, and to show you that Illinois was not shown any partiality, our American Association dates exactly coincide with those of the Illinois State Dental Society.

The innovation of having the general clinics the morning of the first day of our meeting, I believe, is a good one and should be continued in the future. Credit for this idea must go to the illustrious Dr. John R. McCoy, brother of the illustrious Dr. James D. McCoy, both fellow Californians.

I want to call your attention to the splendid work that has been conducted this past year by the Public Relations Committee. Under the able leadership of Dr. T. W. Sorrels and his subcommittee headed by Dr. Frank Nicolai, a great deal has been accomplished. It was my privilege to attend the first meeting of the subcommittee in New York City, and ever since that time I have been in close contact with their endeavors. The amount of work and effort put forth by these men, together with their individual enthusiasm, has produced results which are well worth while and deserving of our wholehearted support.

I urge every member to attend the business session Tuesday night at 8 P.M. in this ballroom. At that time you will hear the detailed report by Dr. Sorrels of the Public Relations Committee. This committee has spent $2500 of your society's money, and each and every one of our members should be vitally interested. There are other matters of extreme importance which will come before our body.

I am hopeful that our constitution and by-laws may be amended in order to facilitate the handling of our association affairs. You will recall that at our last meeting in Kansas City, our constitution and by-laws were so inadequate that the functioning of some of our committees was useless. We are hoping to correct this situation tomorrow night. The ladies are being taken to a theater party so that you will be free to attend the business session.

It is my sad duty to report the names of the following who have been taken by death during the past year:

Percy R. Ashplant
Oscar Busby
Thomas L. Grisamore
J. A. McPhail
H. L. Morehouse
Van A. Stilley
R. B. Van Gieson

At a later session the necrology committee will offer suitable resolutions.

In conclusion, may I take this opportunity to thank you for the honor that you have conferred upon me. I have considered the work involved a privilege and a pleasure. I want to express my sincere gratitude to the chairmen and members of the various elected and appointed committees. Your loyal and wholehearted support during the past year has served the general advancement of our organization. To the incoming officers I offer my congratulations and best wishes. I bespeak for them your continued cooperation.

On almost every side we hear dour predictions concerning the future of private practice. We need not be fearful of the future and what may come if we do our job in this, the present, with the honesty of purpose and sincerity of interest that have been, since the beginning, the symbols of this organization.

636 Church Street

THE CONSTITUTIONAL FACTOR IN SKULL FORM AND DENTAL OCCLUSION

A. LeRoy Johnson, New York, N. Y.

A monograph entitled "The Constitutional Basis of Form and Behavior," by the late Charles R. Stockard, contains an exhaustive detailed discussion of the material presented in this paper. It was Stockard's wish that following the publication of the monograph his co-workers, instrumental in the accumulation of the data upon which he based his more comprehensive discussion, should present short papers pointing out the implications of the work in their respective fields. I, a co-worker, shall therefore endeavor to fulfil this obligation in the field of orthodontics.

CROSSBREEDING contrasted dog types at the Cornell Experimental Farm has produced material which reveals the influence of genetic constitution on skull form and dental occlusion. With this evidence at hand I shall attempt to show that genetic constitution, so long ignored in orthodontic literature, is a most important element in our considerations. It is not my intention to overemphasize the role of genetic factors in the development of dental malocclusion. No student of orthodontics today questions the fact that environmental conditions are often the pervertive influence. My purpose at this time is to show that germinal constitution must also be taken into account in the search for the etiologic factors of malocclusion of the teeth.

The philosophy on which our investigation was based is expressed diagrammatically in Fig. 1. An organism develops by virtue of the interaction of germinal substance with environmental influences. Two complementary phases, genetic and environmental, are involved in all developmental processes. In some instances one, in some instances the other, may be the dominant force, but always both are involved. With the development of the endocrines and the central nervous system a medium of interaction between germinal constitution and external environmental forces is created. Furthermore, evidence indicates an even more intimate correlation between endocrine function and genetic factors. Consequently as the diagram shows, there are three avenues of approach, *A*, *B*, and *C*, in the study of the etiology of dental malocclusion. But in following any one of the three, knowledge and control of the other two are essential. An investigation of the role of germinal constitution, *A*, in dental malocclusion necessitates knowledge and control of internal and external environmental conditions. Otherwise there is no certainty in the isolation and identification of conditions which are of genetic origin. In regard to endocrine or dietetic influence the same is true. An investigation of endocrine influence necessarily involves a knowledge of the genetic constitution of the organism observed, together with its external environment; and in an investigation of dietetic influence, genetic constitution and the nature of the internal environment must be known. Such is the point of view which guided our investigation.

The following discussion deals with, *first*, a general consideration of the dog as suitable material for the study of the influence of germinal constitution on

Read before the Great Lakes Orthodontic Society, Dearborn, Mich., November, 1939.

skull form and dental malocclusion; *second,* a preliminary survey of a great variety of dog skulls which served to define the nature of the material; *third,* the hybridization of contrasted types; *fourth,* effect on skull form of endocrine ablation, calcium deficiency, and surgical removal of osteogenetic cartilages in the spheno-occipital region. In a fifth section, inferences will be drawn, and in a sixth, their bearing upon human genetics and orthodontics will be discussed.

I

Domestic dog breeds show greater variation in skull form than do those of the species of any other natural genus obtainable for experimental investigation. Our modern dog shows exhibit a wide variety of individuals, different in coat color, length of hair, body size, leg length, and especially facial form. Since they are all of one species, cross-mating is possible.

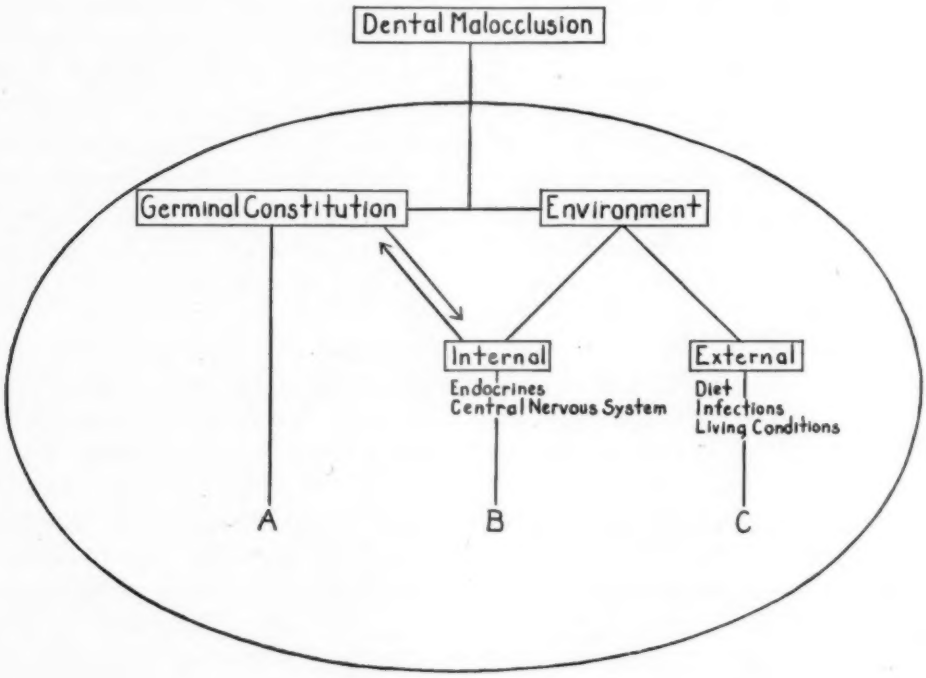

Fig. 1.

Growth deviations in the human face closely resemble those in modern dog breeds (Fig. 2). Compare the maxillary micrognathus condition of the English Bulldog with the so-called Class III case; the Bloodhound with the human acromegalic; and the Pekingese with the cretin. In our hydridization experiments we have produced the "distoclusion" case. In fact, all types of dental malocclusion seen in human beings today are to be found in modern dog breeds or their hybrids (Fig. 3). Because of this similarity, together with the amenability of the dog to experimental routine and the ease with which its living conditions can be controlled, the dog affords excellent material for the investigation of genetic factors in facial conformation and dental malocclusion.

A brief reference to the history of certain modern breeds with accentuated skull modifications and the relation of these modifications to other species will

help to broaden our understanding of certain biologic phenomena with which we are directly concerned in the practice of orthodontics. It will at the same time further emphasize the suitability of the dog for our purpose.

Biologists agree that dogs have come from several wild species. Jones says, "The near relatives of the dog are numerous, and although they are truly wild many are capable of being tamed and most of them will cross with some

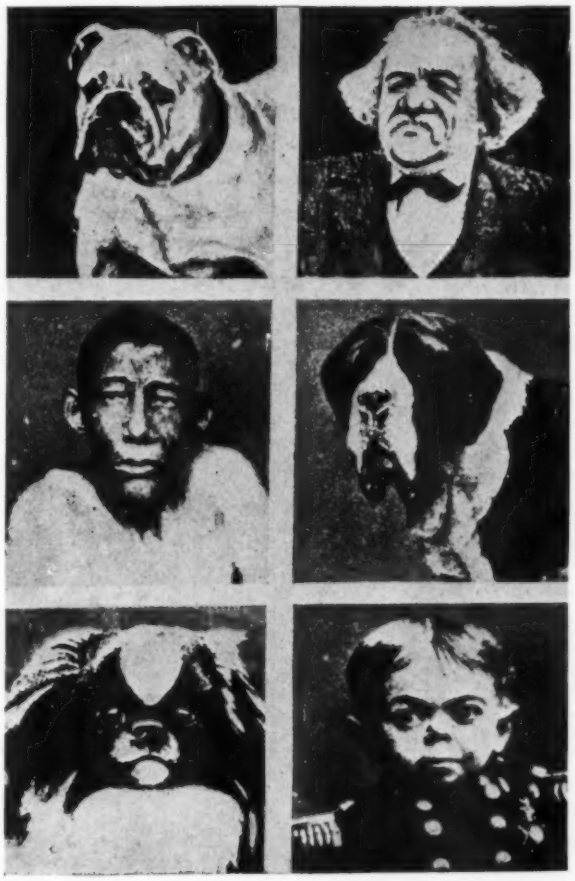

Fig. 2.—(From *Physical Basis of Personality* by C. R. Stockard. Reproduced by courtesy of W. W. Norton and Co., Inc.)

Fig. 3.

breeds of dogs.'"[1] He names the timber wolf of North Europe, the jackal of Europe, Asia, and Africa, and the coyote of North America, in the belief that they have all contributed something to present-day forms. Darwin was of the opinion that domestication of the wild Canidae occurred more or less simultaneously in different parts of the world and that under the influence of domestication new types evolved.[2] Geneticists of today state specifically that the different modern breeds are the result of the artificial selection and preservation of mutations; that mutations occurred in the skulls of the wild Canidae in different parts of the world and have persisted by virtue of man's assistance. The key to their idea is the concept and significance of mutation.

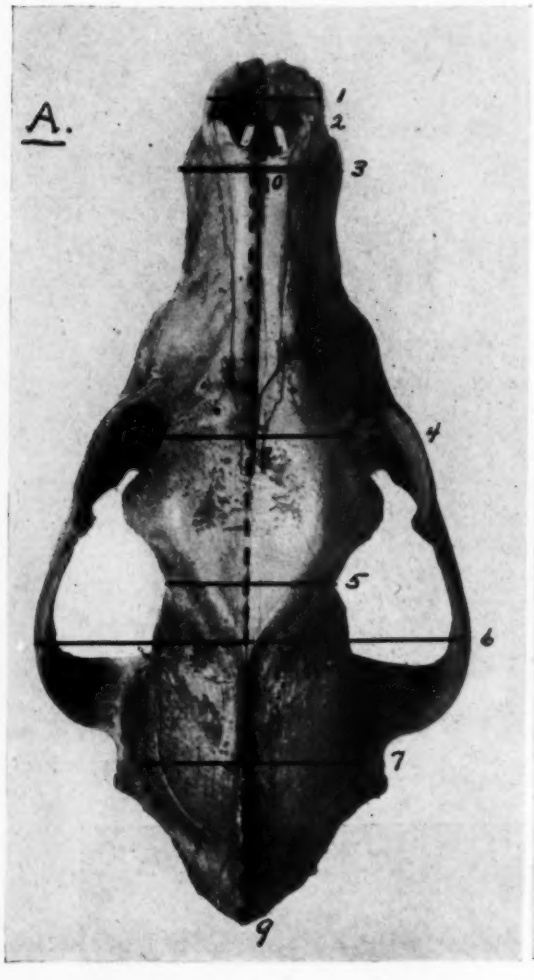

Fig. 4*A*.

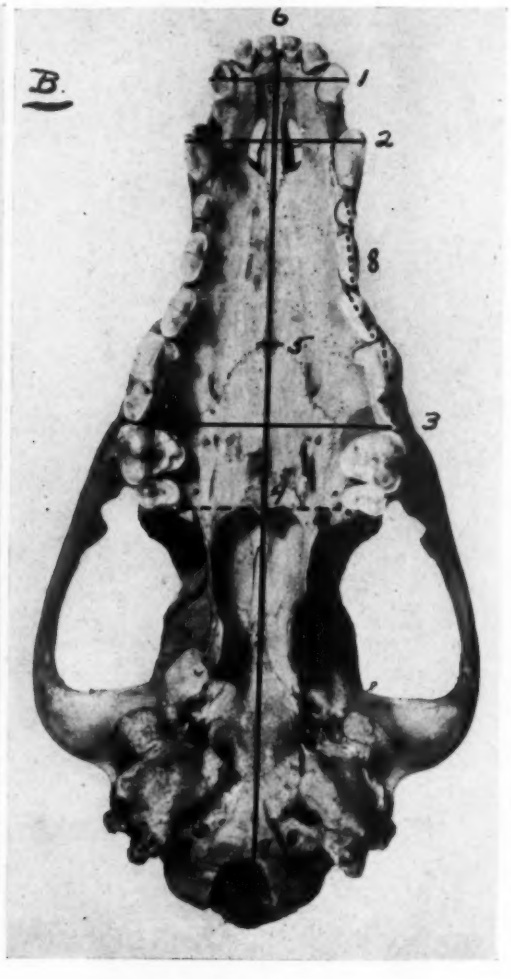

Fig. 4*B*.

Fig. 4*A*.—*1*, Incisal alveolar width. *2*, Nasal width. *3*, Canine alveolar width. *4*, Interorbital width. *5*, Least frontal width. *6*, Zygomatic width. *7*, Parietal width. *8* to *9*, Cranial length. *8* to *10*, Snout length.

Fig. 4*B*.—*1*, Incisal width. *2*, Intercanine width. *3*, Palatal width. *4* to *6*, Total palatal length. *4* to *5*, Horizontal plate of palate bone. *6* to *7*, Total skull length. *8*, Anteroposterior premolar dimension.

Concerning mutation geneticists are not of one mind on all points, yet there is enough common ground to provide us with a fairly clear idea of this phenomenon. Dobzhansky agrees with Stadler that "What in practice is described as a gene mutation is merely the residue left after the elimination of all

classes of hereditary changes for which a mechanical basis is proven."[3] A study of many writers (Jennings, Morgan, Stockard, et al.) shows quite clearly that a mutation is implied when new structural forms appear in a species, forms which cannot be explained on the basis of mechanical changes of the chromosomes. It refers to change in the chemical quality of the genetic complex.

Examples of mutation are seen in leg length of several species. Stockard found that the short leg of the Dachshund, the Bassethound, and the Pekingese is due to a mutation for chondrodystrophy in the epiphyseal cartilages of the long bones of the legs and is inherited as a single factor dominant. Crew reports a ram lamb with short crooked legs, dominant in inheritance, from which, by artificial selection, the Ancon breed was established.[4] Mohr refers to the short legged Dexter breed of cattle.[5] Dunn and Landauer call attention to a similar condition in still another species, the Creeper Fowl.[6] As for the human being, we are all familiar with the short twisted legs of the achondroplasic dwarf. Thus mutation for chondrodystrophy in the epiphyseal cartilages of the legs is not a rare occurrence in several species.

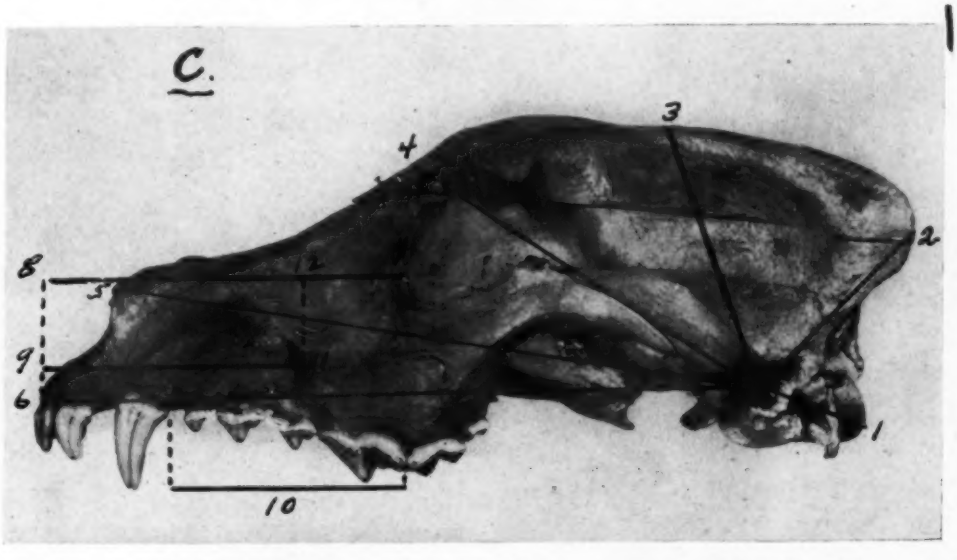

Fig. 4*C*.—*1*, External auditory meatus to condylar process. *2*, External auditory meatus to supraoccipital. *3*, External auditory meatus to bregma. *4*, External auditory meatus to nasion. *5*, External auditory meatus to anterior nasal. *6*, External auditory meatus to superior dental alveolus. 7 to *8*, Orbit to incisal alveolus. *9* to *11*, Orbital foramen to incisal alveolus. 7 to *12*, Orbit to orbital foramen. *10*, Premolar region.

Mutation for bone growth is not, however, confined to the appendicular skeleton. It may occur anywhere in cartilage growth. Parts of the mammalian skull are preformed in cartilage. Chondrification takes place in the basal portions of the occipital, sphenoidal, and ethmoidal regions, and there is reason to believe that mutant changes, similar to those in the leg, occur in these regions of the skull. Among certain dog breeds the skulls show extreme conditions of achondroplasia which are known to be conditions of genetic origin. English and French Bulldogs, Boston Terriers, Brussels Griffons, Pugs, and Pekingese all typify this condition. Babcock and Clausen show cases of extreme achondroplasic conditions in the skull of the monstrous "bulldog" calf, produced by Dexter-with-Dexter matings. Mohr refers to the achondroplasic "bulldog" calf

often found in the Norwegian Telemark breed.[8] Of human beings exhibiting this condition, the cretin with the "dish-face" is usually accepted as the classic example.

"At various times orthodontists have quoted Murk Jansen, a Dutch orthopedic surgeon, to show that the clear-cut modified histogenesis of achondroplasia in the skull is due to the influence of amniotic pressure in the developing fetus. In the light of our investigation of the genetics and development of this condition in dogs, as well as the studies of Landauer on fowls and Knotze on the morphology and histology of this distorted bone growth, such explanations of the histologic processes involved are altogether untenable."[9]

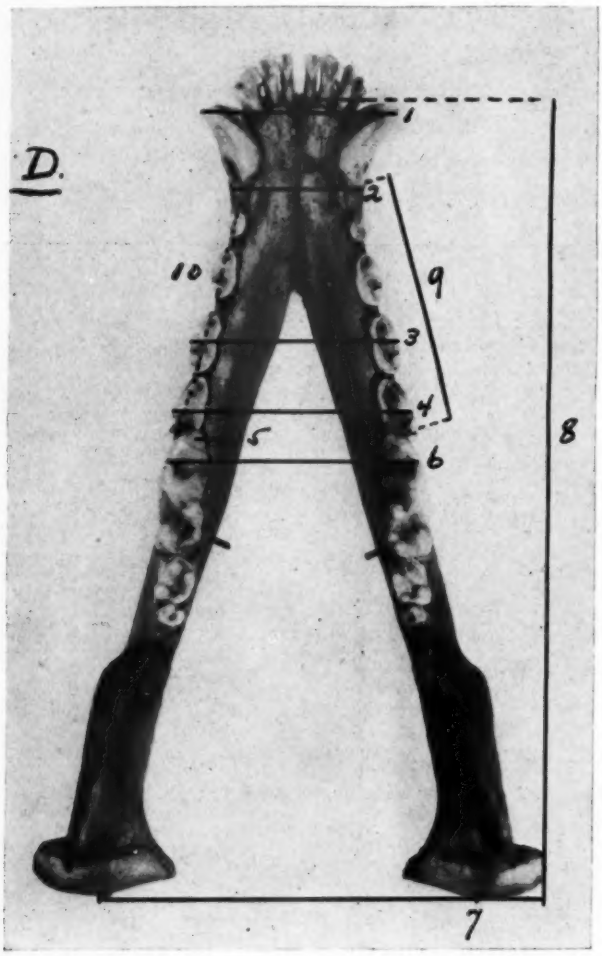

Fig. 4*D*.—*1*, Intercanine width. *2*, Canine alveolar width. *3*, *4*, *6*, Width between third and fourth premolars and first molar. *5*, Mandibular thickness. *7*, Intercondylar width. *8*, Mandibular length. *9*, Premolar region. *10*, Anteroposterior premolar dimensions.

Mutations appear also in other parts of the skull. Any region of the skull or the dentition may express changes in the quality of the germinal elements involved in its development. Such modifications may be small or gross effects in alveolar structure or dental tissue and once occurring may be carried on from generation to generation. An understanding of mutation as it is expressed in the exaggerated facial forms of the dog cannot fail to aid us in detecting mild degrees of the same conditions in otherwise normal individuals.

II

At the outset of our investigation we found in the literature nothing by way of exact information on the growth of the dog skull and no comparative measurements showing characteristic breed differences. We were forced, therefore, to plot numerous dimensions in order to determine which were significantly important in the comparison of the different types. Points on the skull were chosen and a complete series of measurements were recorded from a sample group of skulls of different breeds in order that we might compare them graphically and so determine the areas of type differences and agreements (Fig. 4).

Our sample group consisted of seventy skulls of mature dogs of both sexes. It included German Shepherds, Great Danes, St. Bernards, Salukis, Labrador Huskies, Bassethounds, Foxhounds, Poodles, English and French Bulldogs, Boston Terriers, Brussels Griffons, Dachshunds, Pekingese, Pomeranians, Toys, and a few hybrids. It was, in fact, a collection of as great a variety in skull form as it was possible to get together.

TABLE I

Cranial Width	20 mm.
Least Frontal Width	22 mm.
Auditory Meatus to Bregma	32 mm.
Nasal Width	34 mm.
Interorbital Width	35 mm.
Auditory Meatus to Sup. Occipital	36 mm.
Palatal Width	39 mm.
Intercondylar Width	45 mm.
Auditory Meatus to Nasion	59 mm.
Zygomatic Width	68 mm.
Cranial Length	81 mm.
Palatal Length	86 mm.
Auditory Meatus to Ant. Nasal	131 mm.
Mandibular Length	136 mm.
Auditory Meatus to Sup. Dental	143 mm.
Total Skull Base	233 mm.
Sum of Anteroposterior dimensions of	
Maxillary Premolars	33 mm.
Mandibular Premolars	26 mm.

Numerals indicate the actual difference between extremes.

Fig. 5.

Fig. 5 shows the difference in the extremes of certain actual dimensions of the entire group. With the exception of one width dimension, zygomatic, the anteroposterior dimensions show the greatest fluctuation, the difference in the measurements of the total skull base being 233 mm. As the regions measured approach the cranium, differences diminish; cranial width at the parietal suture showed a fluctuation of only 20 mm. In view of the great variety in skull form and size of the specimens comprising this group, it is interesting to note that the variation in the widths of the palates is but 39 mm. while the palatal lengths show a difference of 86 mm.

The purpose of this preliminary survey was to determine areas of type differences and agreements and to find which parts of the individual skulls vary together. For example: does the length of the skull vary with its width? Do the anteroposterior dimensions of the maxillary teeth vary with the length

of the palate? Stockard's monograph includes a graphic analysis and detailed discussion of this preliminary study which need not be duplicated here. This study reveals the following results of special interest to orthodontists:

First.—In size and form the cranium and the face are independent.

Second.—There is lack of correlation between the upper face and the mandible. A short palate does not necessarily signify a short mandible, nor does a short mandible signify a short palate.

Third.—The size of the dental arch shows a far greater fluctuation than does the size of the teeth.

In view of these conditions it was evident that the hybridization of contrasted dog breeds would throw some light on the role of germinal constitution in dental malocclusion.

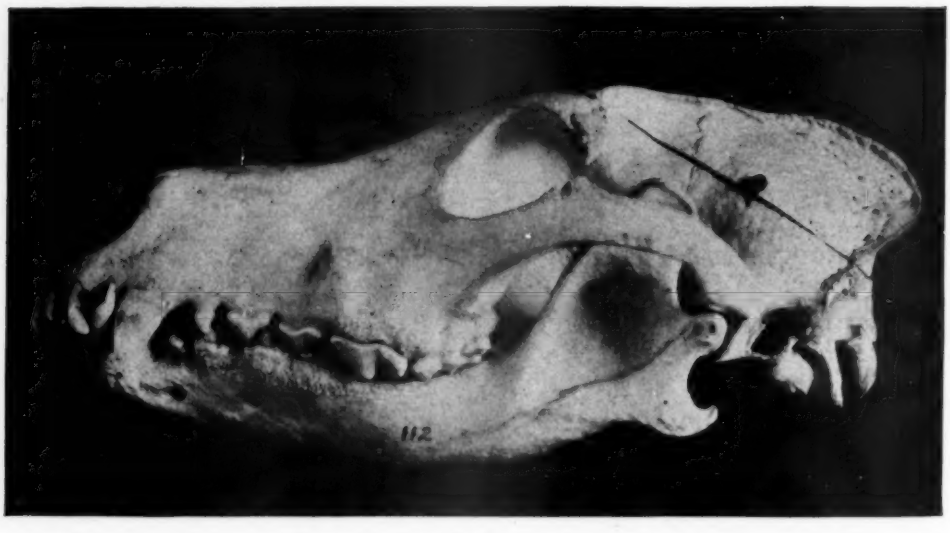

Fig. 6.

In order to identify structural disharmonies in the skulls of certain breeds, a typal pattern as a basis of comparison is necessary. Of all modern dogs, the skull accepted as the normal or standard of the species is the German Shepherd (Fig. 6). Many breeds have departed so widely from the original stem that the ancestral pattern is hardly discernible. But the German Shepherd still exhibits in general the wolf-like skull characteristics of the Canidae. The cranium is low; the snout is long; the horizontal occlusal plane of the teeth is only slightly below the level of the condyles. The posterior border of the hard palate is in the same plane or slightly anterior to the posterior surfaces of the maxillary second molars. The dental formula is I 3/3 c 1/1 pm 4/4 m 2/3 total 42, and thus "lacks only one upper molar of the complete placental dentition."[10] Yet occasionally in some breeds, this third maxillary molar appears today (Fig. 7). In the plate the upper specimen is an F_2 Bassethound by Saluki; the lower, a backcross of F_1 Bassethound-Saluki on a Bassethound. Both specimens descended from a pure-bred Bassethound with three maxillary molars on each side of the dentition. This fact hints, at least, that the genes for the complete placental dentition may not as yet have been entirely eliminated.

While it is not necessary here to present a detailed description of the occlusal relations of the teeth of the dog, certain key relations should be noted. First, the maxillary incisors slightly overlap the mandibular incisors. Second, the mandibular canine projects into the space between the maxillary third incisor and canine. Third, increasing in size from before back, the first three premolars are spaced and are not in occlusal contact with the opposing series. Fourth, the maxillary fourth prelomar and the anterior half of the mandibular first molar are modified in form and in such occlusal relation to serve the shearing, flesh-cutting function, a distinguishing characteristic of carnivor dentition. Fifth, the anterior external cusp of the maxillary first molar occludes with the buccal surface of the trituberculate talon, the posterior half of the mandibular first molar. With these points in mind as normal or standard, atypical conditions of occlusion are obvious.

Fig. 7.

A general distinctive classification of dog breeds is based on skull length in relation to skull width, a long-headed group (dolichocephalic) and a short-headed group (brachycephalic) (Fig. 8). Although any attempt to classify biologic material brings up borderline problems, this general distinction between the long- and short-headed varieties is satisfactory. There are, of course, variations within each group. For example, skulls of the Saluki and several hounds differ from the German Shepherd in the direction of longer, narrower, and more delicately chiseled features. All, however, belong to the long-headed group and possess most of the distinctive features of the ancestral wild type. There is a high degree of harmony in the structural form of the upper face with the mandible and normal dental occlusion the rule. Exceptions to this rule are nearly always limited to comparatively slight variations of incisal occlusal rela-

tions. On the basis of the primitive pattern, the short headed group is undoubtedly abnormal. The bulldogs and freak types show varying degrees of structural modifications, i.e., shorter muzzles, general reduction and distortion of facial features, with disharmony between upper face and mandible, and the invariable presence of dental malocclusion.

In a strict sense, however, there are no pure breeds of dogs. Literally pure line breeding means descendants of two individuals of like germinal constitution, which have not undergone any germinal change because of Mendelian recombination, chromosomal aberrations, or factor mutations.[11] This condition is

Fig. 8.

almost never found in domesticated animals; somewhere back in the line outbreeding has broken the uniformity of the hereditary constitution. Seldom if ever are the germinal factors of a domesticated animal homozygous. Nevertheless, many of our so-called pure breeds of dogs have been inbred for so many generations that the genetic base of certain characters, not the animal as a whole, appears to be sufficiently homozygous to anticipate these characters with a high degree of certainty.

As a subject of genetic analysis, the skull is much more complex than the extremities. Certain factors of the genetic pattern influence certain parts or regions of the skull in either a dominant or a recessive manner. Because of this complexity there still remains a great deal to be done before a complete understanding of the influence of germinal constitution on skull form can be presented. We have based our studies on the comparison of actual dimensions and the computation of indices of localized regions. (Fig. 9.) Approximately a thousand skulls have been measured and recorded. However crude the future may prove our methods to be, they are sufficiently effective to show, in a general way, the necessity of considering genetic constitution in the study of structural anomalies of the skull.

DERIVATION OF INDICES

Cranial Index	Cranial Width / Cranial Length
Skull Index	Zygomatic Width / Skull Base
Palatal Index	Palatal Width / Palatal Length
Snout Index	Maxillary Canine Width / Snout Length
Upper Facial Index	Nasal Length / Palatal Width
Breadth-Height Index	Bregmatic Height / Cranial Width
%Maxillary Premolar to Premolar Region	Sum Ant.-Post. Dimensions Max. PreMs. / Premolar Region. Length
%Mandibular Premolar to Premolar Region	Sum Ant.-Post. Dimensions Mand. PreMs. / Mandibular Region. Length
Mandibular Index	Intercondylar Width / Condyle to Symphysis

Fig. 9.

III

As before stated, in order to determine the role of genetic factors in the development of skull and dental malformations, it is necessary to control external living conditions and to study the nature of endocrine function and nervous reactions of the animals used. These requirements were met through the following means: The living conditions were as uniform as it was possible to make them. Food, kennel, and climate were the same for all. At autopsy

the endocrine glands were removed and made the subject of special histologic study.[12] The character of the central nervous system of the different breeds was revealed in the study of the Conditioned Reflex by James and Anderson.[13]

In these hybridization experiments it made no difference which parent was male or female. There appears to be no sex-linkage in the characters with which we are here concerned.

The following crosses have been selected to illustrate the results of our experiments:

In the upper row, left center, Fig. 10, is the pure bred Bassethound skull. In general form it closely approximates the normal ancestral type exemplified by the German Shepherd. It is dolichocephalic, with cranium low in relation to the face. Maxillary and mandibular structures are patterned harmoniously to each other, and there is a full complement of teeth in normal occlusion and alignment. At the left the ventral aspect shows the posterior edge of the palate even with the posterior surface of the maxillary second molars. A line drawn posteriorly from the canines to the second molars curves slightly outward from the median line in the region of the fourth premolar and first molar.

In striking contrast is the skull of the English Bulldog, upper right center. Although certain Bulldog characteristics are occasionally seen in the dolichocephalic group, the better specimens, championship stock, of which this is one, are markedly brachycephalic. The cranium is high, well above the face, giving the face a comparatively vertical angle. The nose is extremely short and the mandible prognathus. From the ventral view of the skull at the right, a line drawn posteriorly from canines to the second molar shows a decided curve away from the median line in the region of the maxillary fourth premolar and first molar. The posterior edge of the palate is posterior to the second molars. Young animals usually have a full complement of teeth but the occlusion is poor. The maxillary teeth are badly crowded with premolars rotated away from their normal position.

The mandible of the Bassethound conforms to the ancestral pattern, but the English Bulldog mandible departs from it. The horizontal dental occlusal plane of the Bulldog is curved with the convexity downward and is relatively far below the condyles; and in striking contrast to the Bassethound, the incisors and canines flare outward to a nearly horizontal position.

Below the pure breds are two skulls of the F_1 generation. These are, genetically, 50 per cent each Bassethound and English Bulldog. In general form they are similar and might easily be considered distorted Bassethound skulls. As we shall see in the analysis of larger numbers of this cross which follows, the bulldog type skull is on the whole recessive in inheritance. Yet the undershot condition and the deeper depression at the nasion in the skulls of the F_1 generation suggest that certain parts of the bulldog skulls are dominant in this hybrid. All F_1's of this cross in our possession show normal occlusal relations of the molars with the maxillary incisors and canines posterior to their normal relation with the corresponding teeth of the mandible.

The lower row of six skulls is derived from cross-mating two of the first hybrid generation. Passing from right to left is a gradual modification in form from the Bassethound type toward the Bulldog. That this change is the result

of a reduction in the anteroposterior dimensions of the maxillary structure is evident by comparison of the mandibles, which are strikingly similar. The hound type of mandible prevails. Even at the extreme left where the upper face approximates the Bulldog, the mandible in form and the dentition in arrangement are nearer that of the hound than of the Bulldog.

A very important point for the orthodontist to note is that as the snout becomes shorter, the teeth are not correspondingly reduced in size. They are crowded, rotated, and faulty in alignment and occlusion. Where the snout is short, the teeth are too large for the jaw.

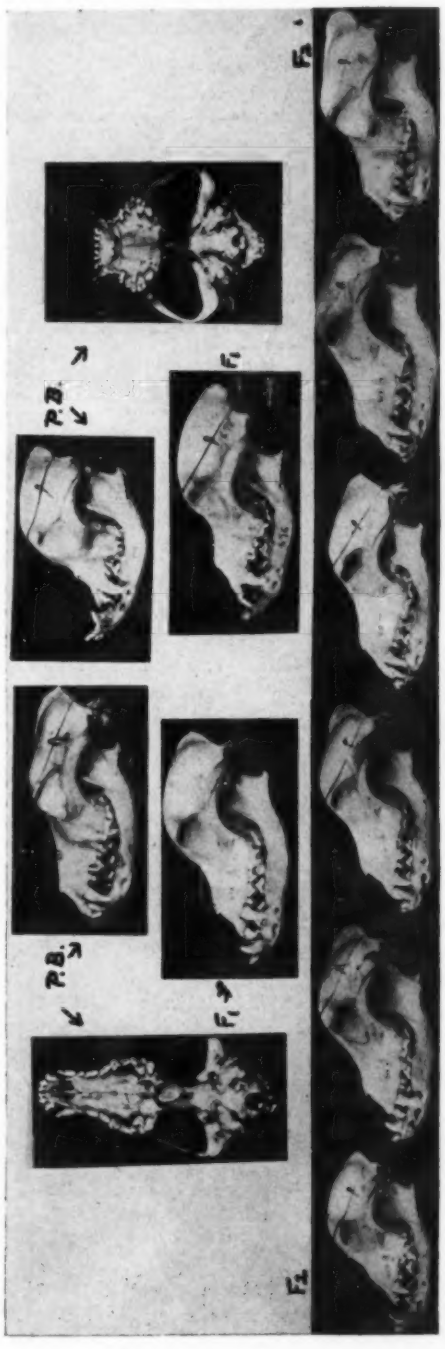

Fig. 10.—Basset hound by English Bulldog.

There is no significant difference in cranial and breadth-height indices which are obtained from cranial and near cranial dimensions (Fig. 11). But indices of the upper face, i.e., skull, palatal, snout, and upper facial, show decided contrast between the pure breds. All upper facial indices of the F_1 group are nearer the long-snouted, more primitive type of the Bassethound than they are to the short-snouted Bulldog. A majority of the characters of the Bulldog skull are undoubtedly recessive in this cross. In the F_2 generation, which shows a greater difference in structural forms and relations than the parent F_1's, the general trend is toward the hound rather than the bulldog. At one end of the scale an F_2 skull will be found so nearly like the Bassethound that it might easily be mistaken for it, but at the other end the pure Bulldog type is never quite duplicated in this generation.

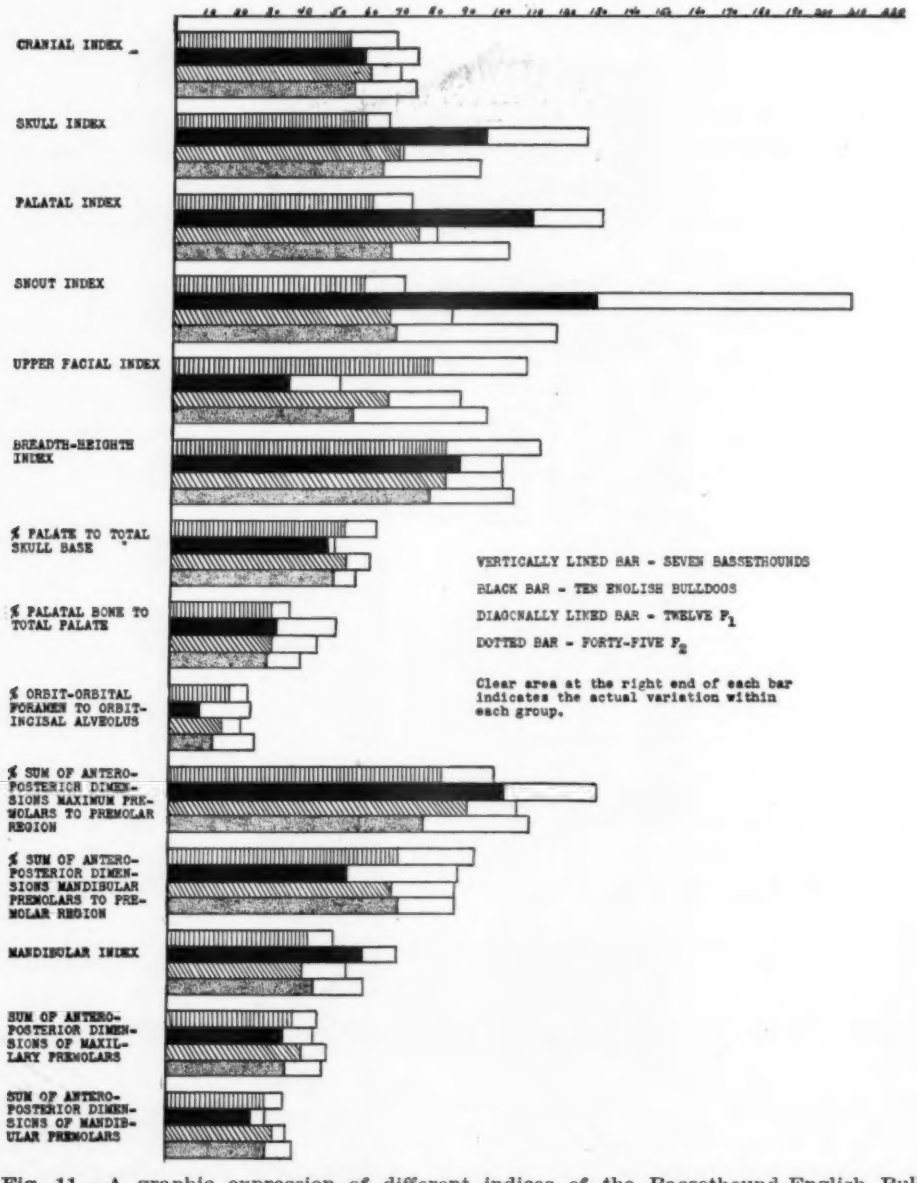

Fig. 11.—A graphic expression of different indices of the Bassethound-English Bulldog cross for a comparative study of the pure breds and their hybrids in larger numbers derived from several matings.

Between the three generations, relations of size of teeth to jaw length in the maxilla do not show the degree of difference that is seen in a comparison of the indices of the bones of the face. This is due to the fact that the index, an expression of form, is not strictly comparable to this percentage, which is a ratio of single dimensions. Counting downward from the top of the graph, the tenth group shows the amount of space in the premolar region of the maxilla that is occupied by the premolar teeth. All pure bred Bassethounds show 100 per cent or less. The premolars do not overlap each other and in the majority of instances they are spaced in some degree. The English Bulldog shows just

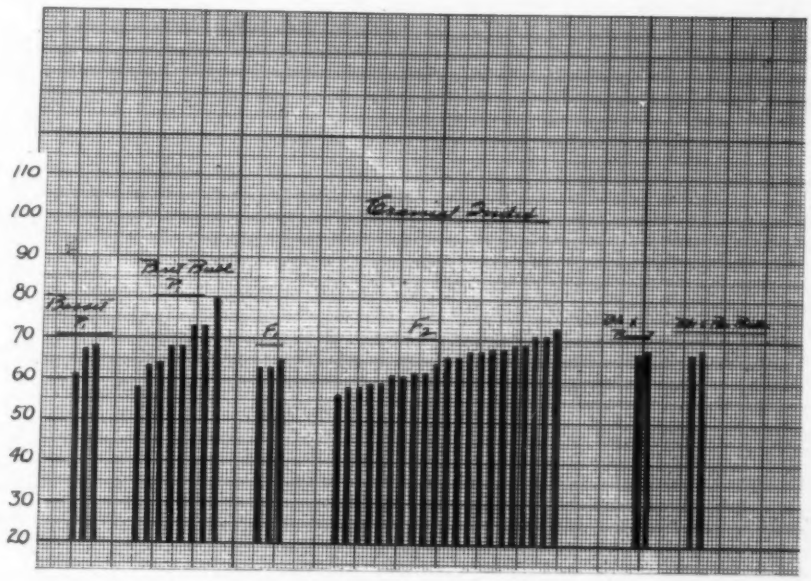

Fig. 12*A*.

Figs. 12 *A-E*.—A study of cranial indices (*A*), skull indices (*B*), snout indices (*C*), palatal indices (*D*), and mandibular indices (*E*), showing the distribution of individuals in different generations.

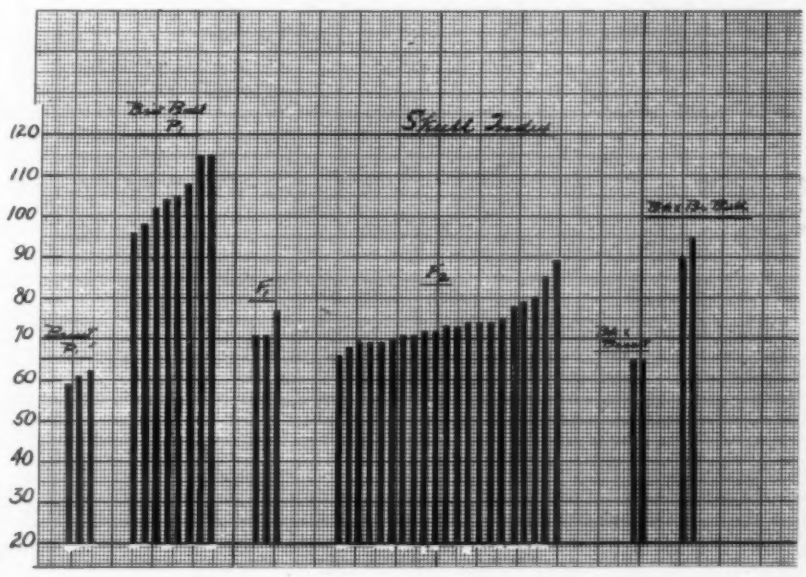

Fig. 12*B*.

the reverse condition. In all ten of these skulls the premolars are too large for the premolar region. When all premolars are present they are nearly always rotated and out of line. The F_1 generation fluctuates around 100 per cent but tends toward the long-nosed Bassethound parent rather than the short-nosed English Bulldog. The F_2 generation shows a greater variation but as a whole this group tends toward the Bassethound rather than the Bulldog.

In regard to the relation of the mandibular premolars to the premolar section of the jaw, there is no significant difference in the three generations. Although the graph shows that the Bulldog teeth in certain specimens are more widely spaced, i.e., they occupy a smaller part of the jaw than in the Bassethound, and the average for the group is lower, the fluctuation is so great among these so-called pure breds that the mandibular conditions of all three generations are nearly alike and the apparent differences are insignificant.

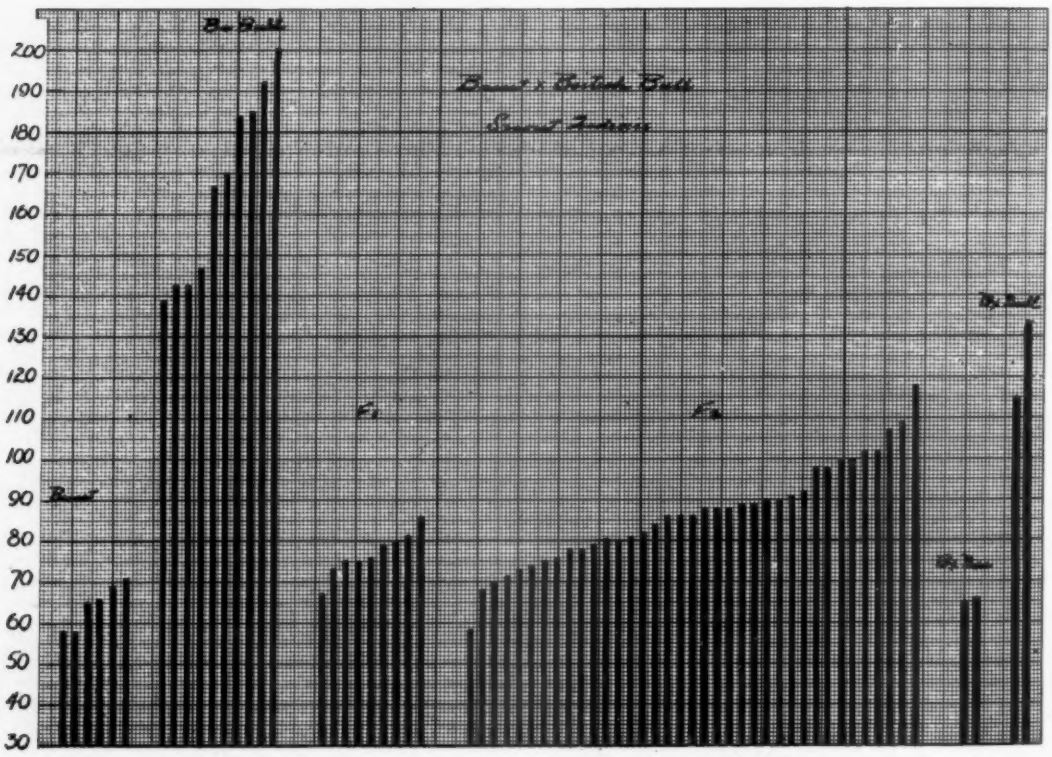

Fig. 12*C*.

At the lower part of the graph are two groups of comparative studies in size as expressed in anteroposterior dimensions of premolars. Both maxillary and mandibular premolars of the Bulldog are slightly smaller than those of the Bassethound, but the difference is negligible. Thus there can be no doubt that differences observed in groups 10 and 11 are due to differences in jaw dimensions rather than to differences in the size of the teeth.

Each vertical represents the cranial index (*A*) of one dog (Fig. 12). At the left are the indices of three Bassethounds. The next group to the right are the indices of eight English Bulldogs and then three of the F_1 generation and

twenty-one of the F_2. At the extreme right are two groups of two dogs each, showing the indices of the back-crosses. This study of the cranial indices is of interest when compared with the skull indices (*B*) in the lower half of the same figure, also the snout (*C*), palatal (*D*), and mandibular (*E*) indices. The differences of cranial indices in the three generations are not significant, but *B*, *C*, and *D* are clearly contrasted following the expected distribution according to Mendel. Back-crossing, the mating of an F_1 with each of the pure bred parents,

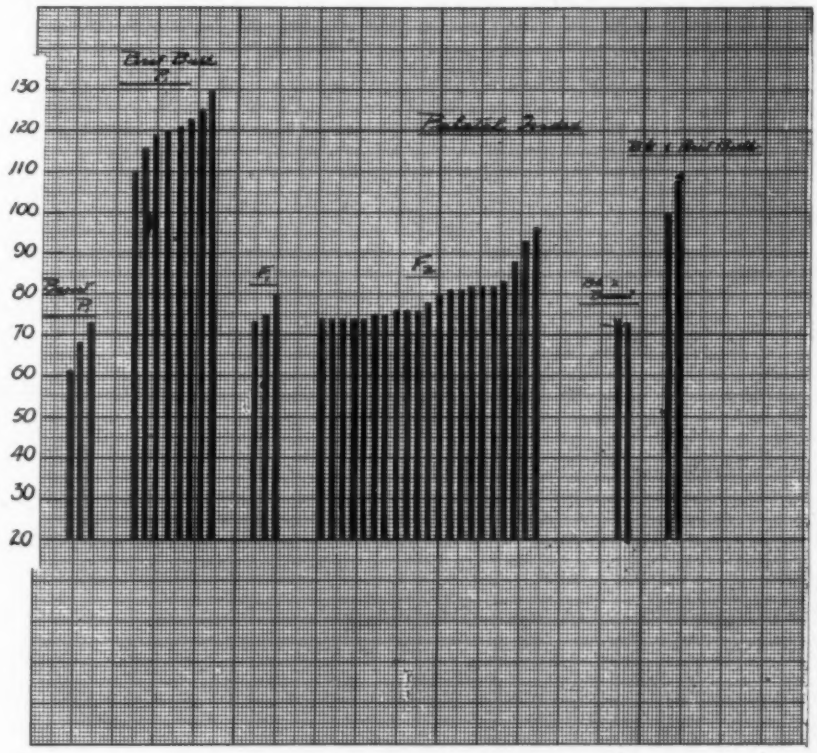

Fig. 12*D*.

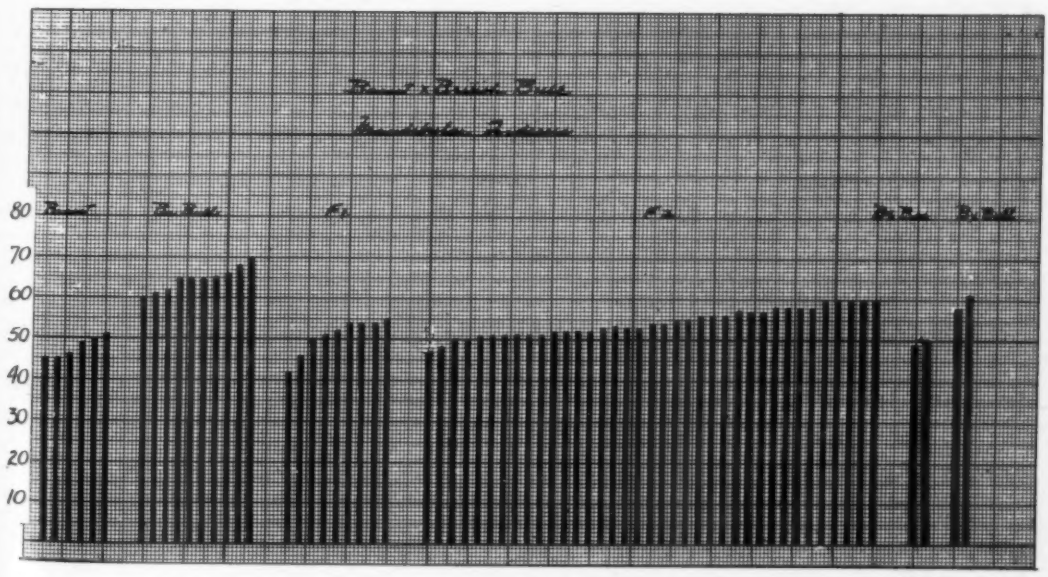

Fig. 12*E*.

produces striking evidence of the influence of genetic constitution on skull form. The F_2 generation shows great fluctuation, ranging from a skull form comparable to that of the Bassethound toward that of the English Bulldog, though never quite duplicating the latter. Mandibular indices do not show the extreme differences seen in those of the upper face. The high index of the bulldog mandible is probably due more to the great intercondylar width, an adjustment to the achondroplasic skull base, than to a peculiar form of the mandible as a whole resulting from genetic mutation. Genes for the primitive type mandible, in this instance that of the Bassethound, are certainly dominant in all hybrids of this cross.

Graphs of this kind of all indices and of many lineal dimensions show the same uniformity of individual distribution in the different generations of the Bassethound-English Bulldog cross. In this cross the evidence is convincing that genetic constitution is an important factor in the development of skull form and dental occlusion.

Fig. 13 shows the difference in structural form of the achondroplasic (left) from the normal dog skull (right). In the latter the two osteogenetic cartilages are clearly seen, the anterior dividing the body of the sphenoid into anterior and posterior segments. The posterior cartilage is comparable to the basi-occipital cartilage present in the human skull. When normal growth ceases, these cartilages disappear. A condition of chondrodystrophy in this region means premature arrest of growth with resultant structural distortion of all parts preformed in cartilage. As these modified areas constitute the basal structures of the skull, the superstructure, i.e., the upper face, is inevitably modified in form. This is particularly evident in the anteroposterior dimension.

Chondrodystrophy of the chondrocranium appears to be related to genetic constitution. In the center of the body of the sphenoid of the English Bulldog is a canal running in a vertical direction from the nasopharynx to the cranial cavity at the sella turcica. The pituitary which lies in the sella turcica is abnormal in this breed. The anterior lobe and pars intermedia are developed from a diverticulum in the roof of the primitive mouth (the stomodeum).[14] This diverticulum, Rathke's pouch, pushes upward into the cranium. All specimens do not exhibit a canal so clearly defined as does the specimen here shown, yet a great many have openings of various sizes, indicating that complete metamorphosis has not occurred in the development of the anterior lobe of the pituitary. Pharyngeal tissue is still continuous with the anterior lobe. The problem of achondroplasia is complex. Genetic factors are definitely expressed in the bulldog skull. Although the hormones of the abnormal pituitary are in the blood stream, the skeletal modifications of this animal are limited mainly to the head and tail. On the basis of our findings, I venture the summary statement that hormones of defective endocrines circulating in the blood stream are localized in their effect by the genetic constitution of a part. Linkage between genetic constitution and endocrine function in certain skull types is unquestionably a fact which demands further study, inasmuch as it bears directly on so many clinical problems in orthodontics.

From left to right in Fig. 14, the first two skulls are normal; the third is the skull of a cretin. Below the skulls are drawings from measurements made at the median sagittal planes. Compare the curves *G—H—J—K*. In this region the two normal skulls, though of different types, are very much alike. The cretin shows a highly arched curve with a foramen magnum (*G—H*) approaching a vertical position. At the base of this skull the chondrodystrophy characteristic of the achrondroplasic dwarf has resulted in a marked modification in structural form. It is this condition which gives the "dish-faced"

Fig. 13.—Median sagittal sections of the skull of the English Bulldog and the Bassethound.

appearance to the profile of the cretin. There is reason to believe that we see degrees of this condition in orthodontic practice. Certain so-called Class III cases are in all probability maxillary micrognathus conditions resulting from chondrodystrophy in the growth cartilage at the base of the skull rather than from an overgrowth of the mandible.

The black areas in the nasopharyngeal regions of the two skulls outline the structural formation of these regions (Fig. 15). A comparison of these two

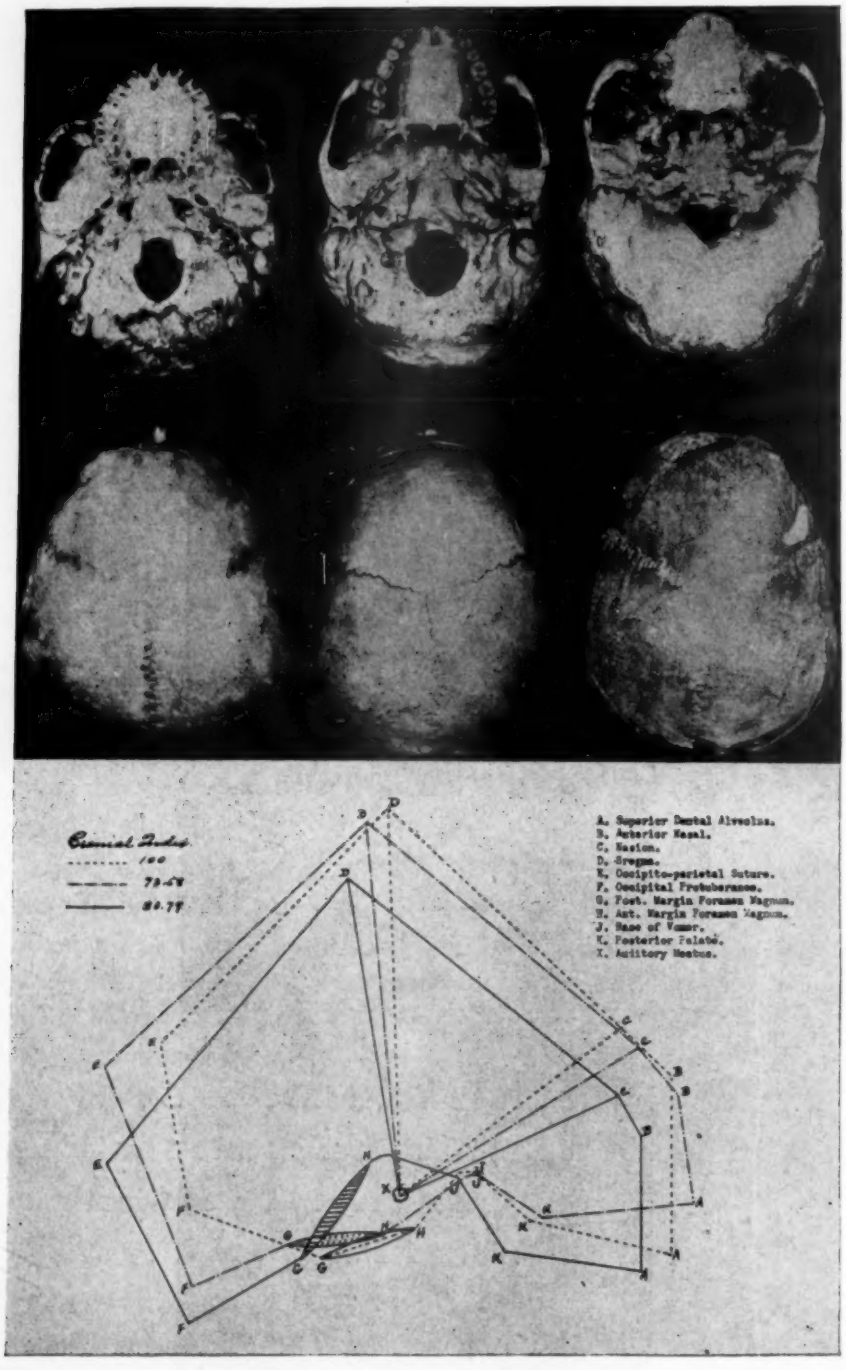

Fig. 14.—Three human skulls with cranial indices, from left to right, of 100; 72.58; and 80.79.

shows a correlation between skull form and dental occlusion and suggests a chondrodystrophy in the right hand specimen.

The Dachshund skull (upper left center, Fig. 16) is the same type as that of the Bassethound, varying but little in form and size. It is built on the primitive, ancestral Canidae pattern with the distinguishing characteristics of the long-muzzled group. There is, however, very little if any spacing between the maxillary premolars. The mandibular premolars are spaced.

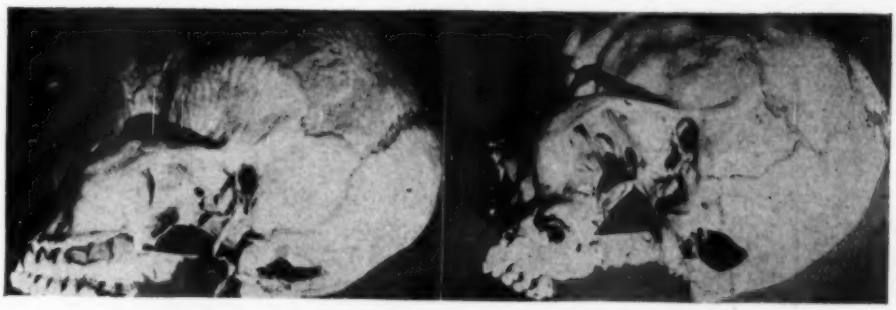

Fig. 15.—Two human skulls: left, normal occlusion; right, Class III.

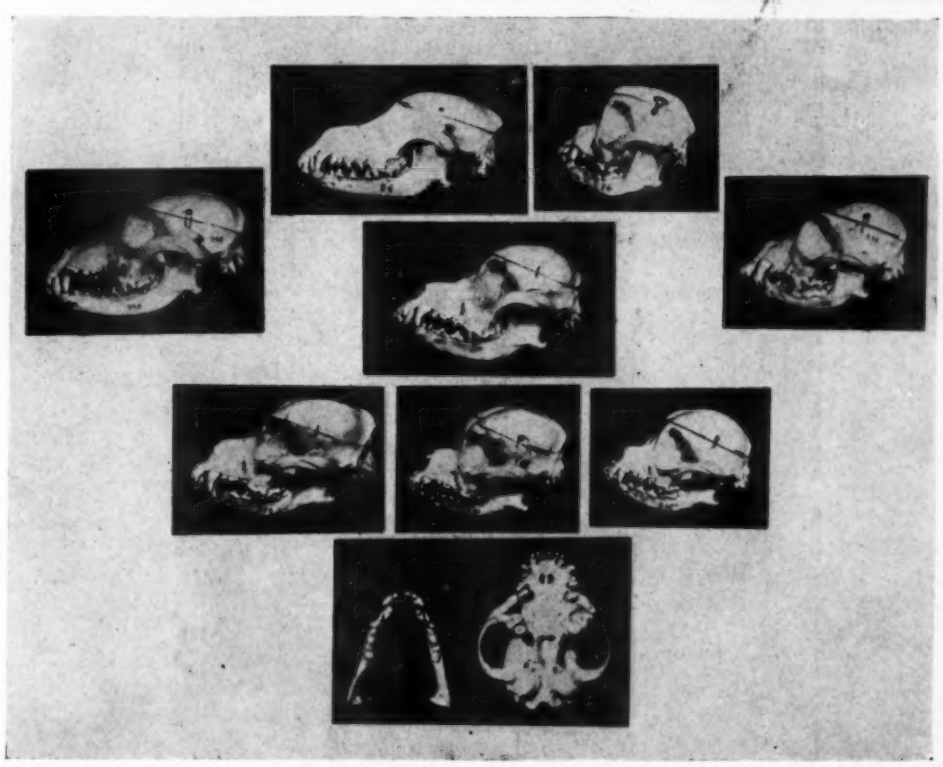

Fig. 16.—Pure bred Dachshund by pure bred Boston Terrier.

The Boston Terrier skull (upper right center) is unquestionably of the short skull variety. Most details of the upper face are comparable to the English Bulldog. But unlike the English Bulldog, the mandible of the Boston Terrier is short antero-posteriorly, giving an end-to-end occlusal relation of the incisors. In fact, this occlusion of the incisors is practically the only definite

occlusion of the entire dentition, for both the maxillary and mandibular teeth are badly crowded and out of alignment, as shown in the ventral view at the bottom of the figure. In comparison with the English Bulldog the dental occlusion is more defective. Directly beneath the two pure breds is one F_1 and the three below this are litter mates of the F_2 generation. The extreme right and left skulls near the top are back-crosses. The left is three-quarters Dachshund and one-quarter Boston Terrier, while the right is one-quarter Dachshund and three-quarters Boston Terrier. These specimens are obtained by mating the F_1 with the pure bred Dachshund and the Boston Terrier. The back-cross on

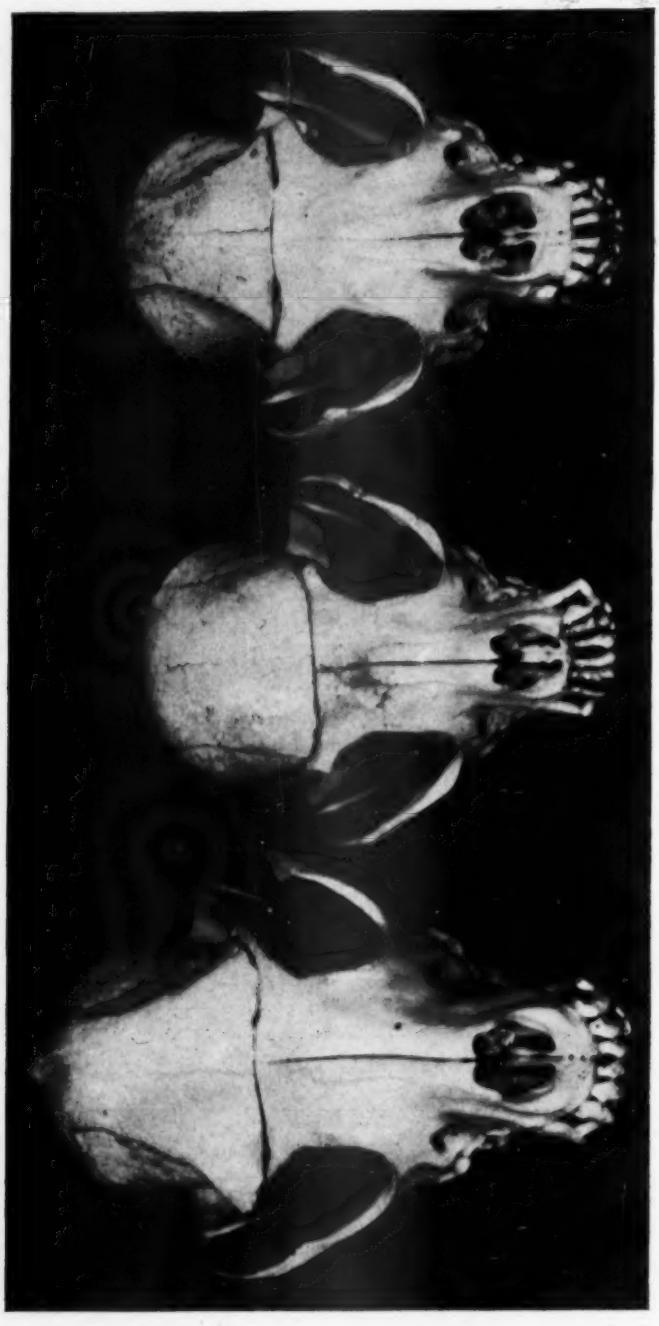

Fig. 17.—Dachshund by Boston Terrier F_2 litter mates.

the Dachshund produces a skull of the Dachshund type, but in the Boston Terrier backcross the upper face is neither so short as the Boston Terrier nor so long as the Dachshund; it is more nearly intermediate between the two. Its mandible, although markedly like that of the Boston Terrier, shows a posterior occlusal relation of the mandibular teeth. Hence in this instance a germinal constitution of a mixture of Dachshund and Boston Terrier genes with a majority of the latter produced a "distoclusion" case.

The dorsal view of Fig. 17 shows a difference in size of three litter mates of the F_2 generation, and also the difference in alveolar structure surrounding the roots of the incisors. It should be remembered that these three dogs had the same parents and grandparents and were reared under the same living conditions.

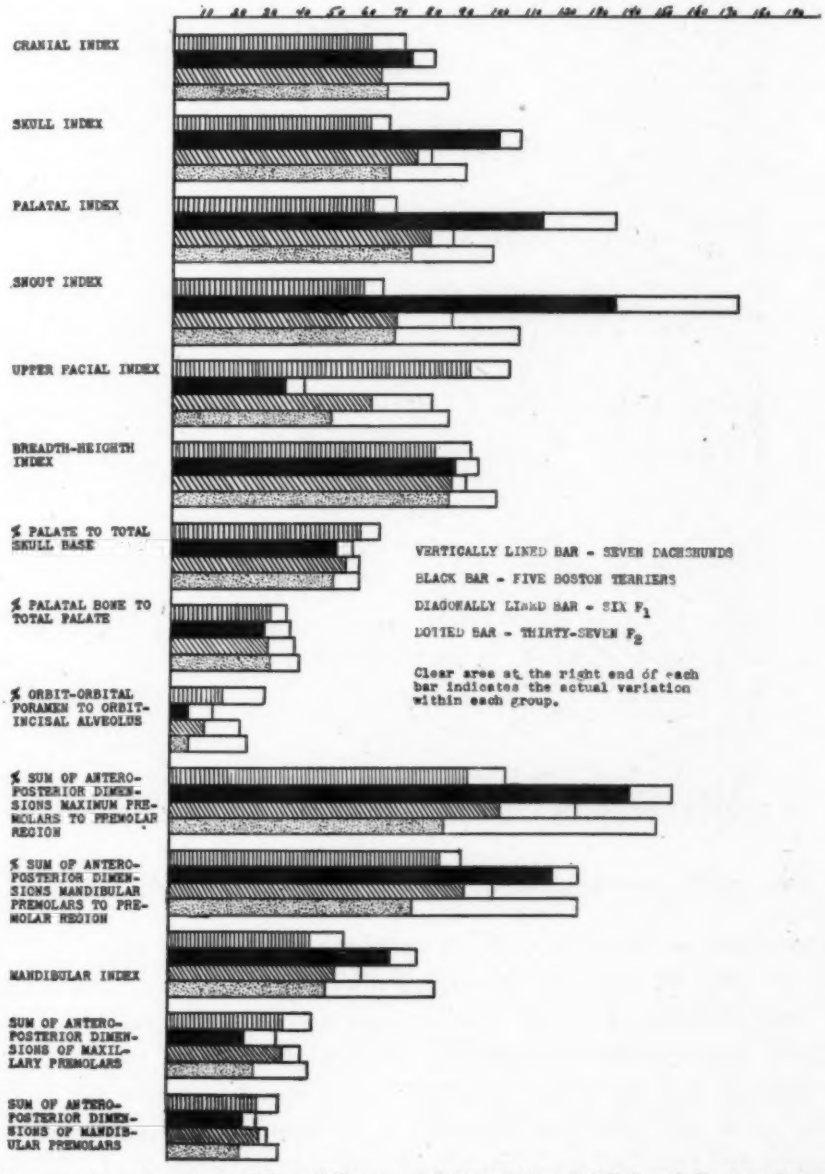

Fig. 18.—A graphic expression of the different indices of the Dachshund-Boston Terrier cross.

In Fig. 18 for the upper face the variations in form of structure follow practically the same line of distribution as the Bassethound-English Bulldog cross. (See Fig. 11.) The genetic factors of the Boston Terrier are more definitely recessive. This is apparent in the character of the fluctuation in the F_2 generation. Although reaching out toward the Boston Terrier, the indices for the upper face do not approximate it as closely as the F_2 group of the Bassethound-English Bulldog cross approach the English Bulldog.

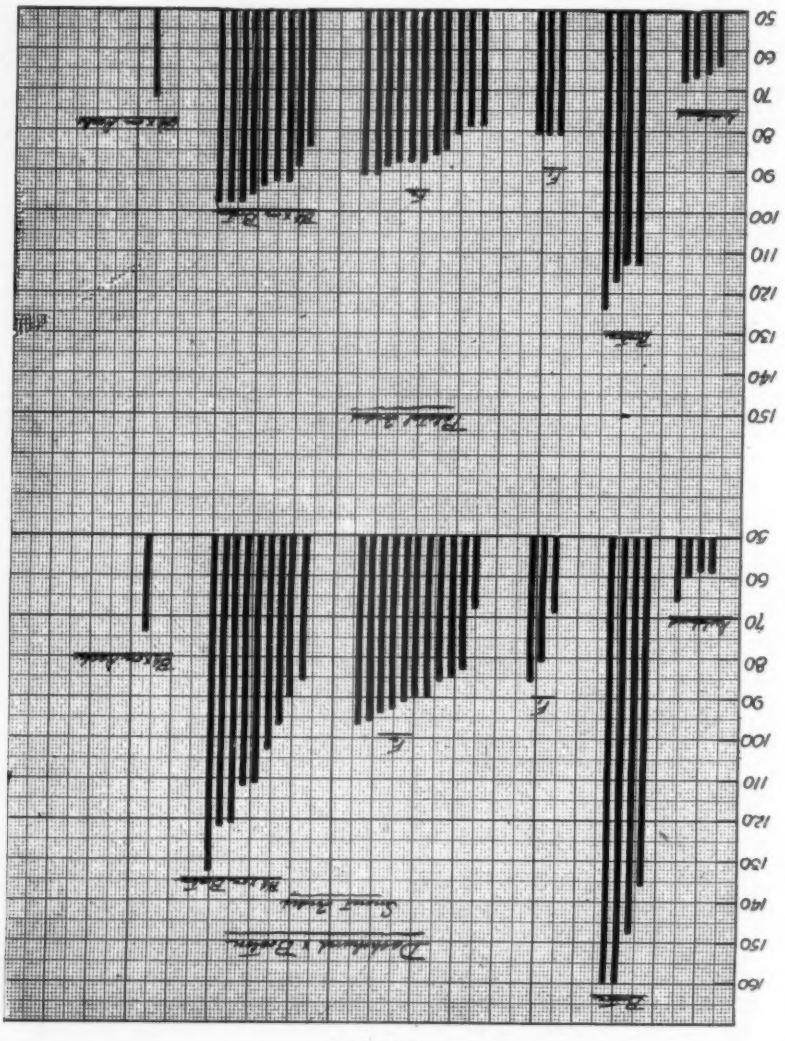

Fig. 19*A*.

Figs. 19 *A*. and *B*.—The distribution of individuals from a Dachshund-Boston Terrier mating.

The tenth group from the top brings out an interesting fact in the relation of the sum of the anteroposterior dimensions of the premolars to the premolar region of the maxilla. Percentages of the seven pure bred Dachshunds show that the premolars practically fill this region. In some cases there is a little spacing and in others a slight crowding. In the case of the Boston Terrier, even when the first premolar is missing, as is often the case, the other premolars are crowded and rotated out of normal alignment. The bar of the

F_2 generation shows that there are instances when the spacing of the premolars is greater than that of the pure bred Dachshunds, the grandparents, and also some equally as crowded as the pure bred Boston Terrier. The next group below illustrates the relation of the premolars to the premolar regions of the mandible. Both groups of pure breds show less variation in this particular than do the Bassethounds and English Bulldogs. This is also true of the F_1 generation. In the F_2 generation the differences are greater, extending from wider spacing than is ever found in the pure bred Dachshund to as crowded

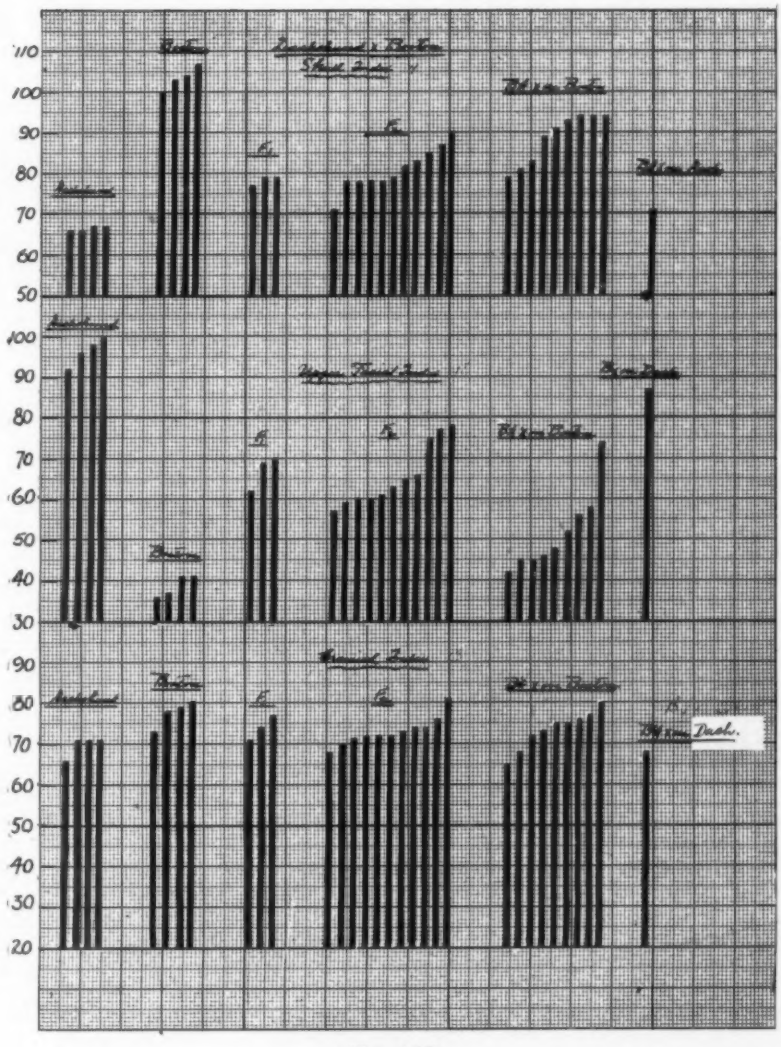

Fig. 19*B*.

a condition as is found among the pure bred Boston Terriers. This, together with a great fluctuation in the mandibular indices in the F_2 generation of the next group below, is indicative of the peculiar genetic quality of the Boston Terrier mandible. A comparative study of the actual dimensions antero-posteriorly of the premolar teeth of the different generations is presented in the two lower groups.

Although only the snout, palatal, skull, upper facial and cranial indices are shown in Fig. 19, the same method of study of all indices and dimensions

reveals a like uniformity of distribution. Though varying in degree of fluctuation they present much the same picture as the Bassethound-English Bulldog cross.

Number 1 in Fig. 20 is the pure bred Dachshund. Number 2, the Pekingese, is one of the most distorted skulls of any dog breed. With the Brussels Griffon it is so unlike the ancestral type that it might easily be taken for another species. Anteroposteriorly it is so abbreviated that the face approaches the vertical angle of the anthropoids. In contrast to the English Bulldog the

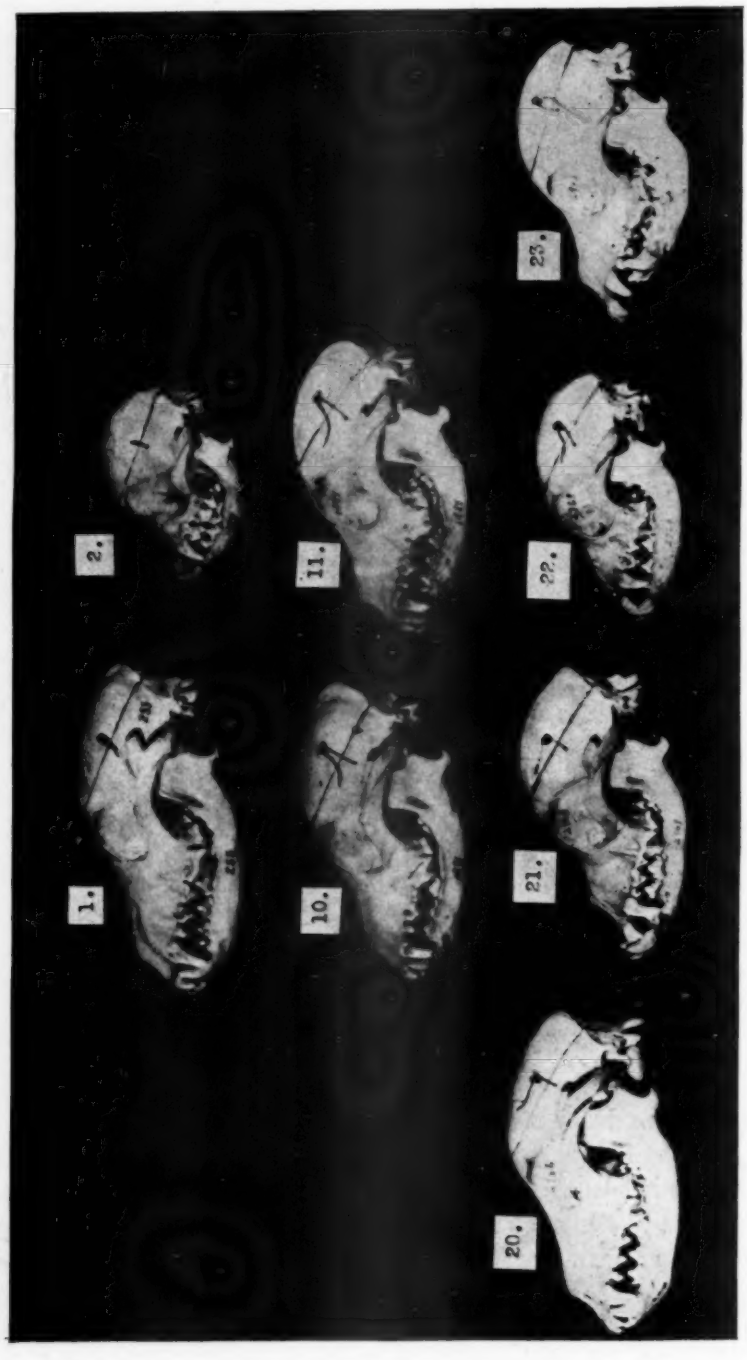

Fig. 20.—Dachshund by Pekingese.

mandible does not extend far beyond the maxilla, but is short like that of the Boston Terrier.

The dentition is very defective. There is seldom a full complement of teeth. Malocclusion and abnormal alignment are the rule. Incisal occlusion is either end-to-end or the mandibular incisors are slightly anterior. Occasionally the mandibular second molar is impacted in much the same manner as the human mandibular third molar.

Numbers 10 and 11 are litter mates of this Dachshund-Pekingese cross. Others of the F_1 generation show a greater difference in skull form than these two litter mates, although all tend definitely toward the Dachshund type. The third row, Numbers 20, 21, 22, 23, the F_2 generation, are litter mates from a

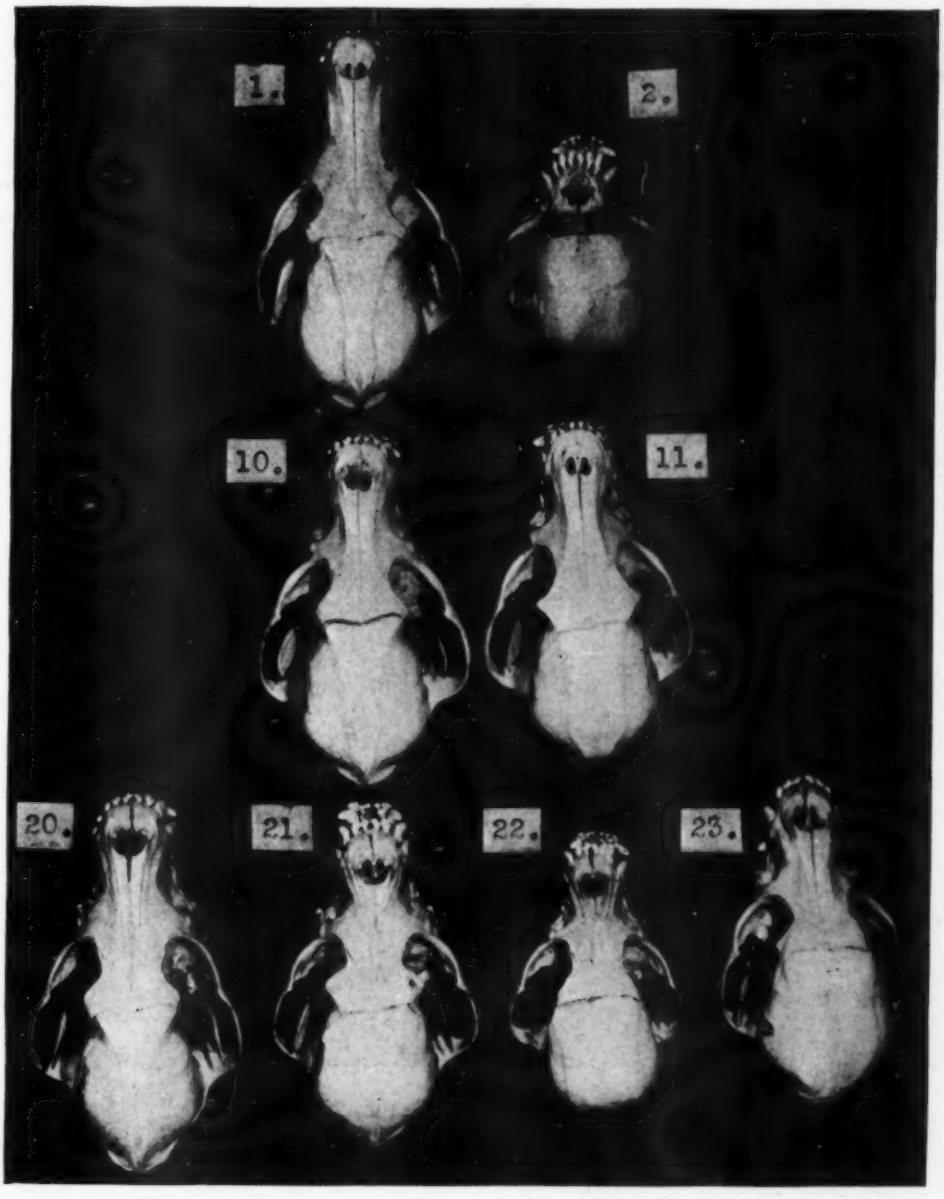

Fig. 21.—Dorsal view of cranium and upper face of Dachshund-Pekingese cross.

cross of Numbers 10 and 11. Number 20 might readily be taken for a pure bred Dachshund in both skull form and dental conditions. Numbers 21 and 22 are intermediate between the pure bred grandparents, while the upper face of Number 23 tends toward the Dachshund and the mandible even more decidedly toward the Pekingese. In Number 23 there is practically no resemblance to normal occlusion of the teeth, and the relation of the mandible to maxilla is comparable to the "distoclusion" case frequently met in orthodontic practice (Figs. 21 and 22).

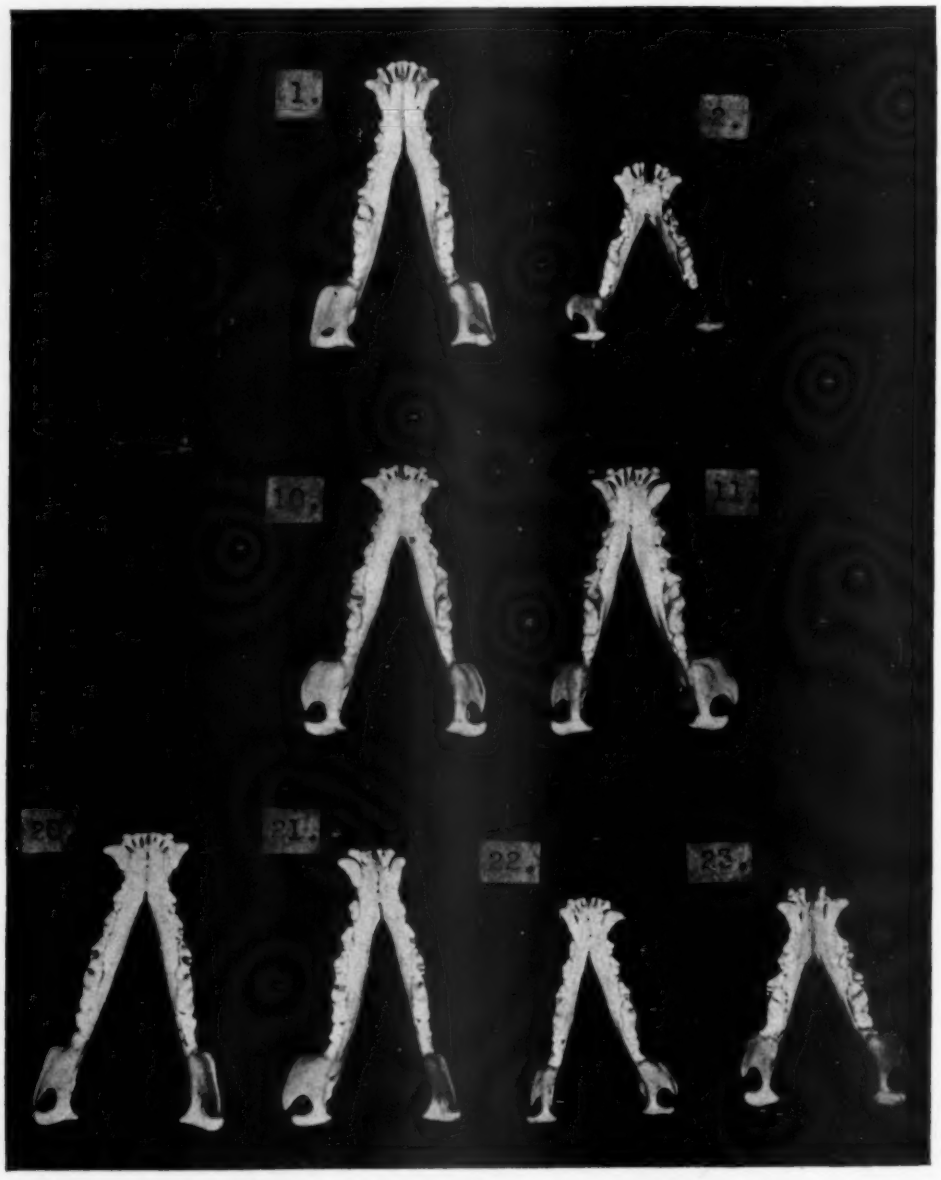

Fig. 22.—Mandibles of the same animals as in Fig. 21.

When at all relaxed it was not possible for this dog to keep the tongue within the oral cavity (Fig. 23). Both upper face and mandible were short like the Pekingese while the tongue was long and narrow like the Dachshund.

That both jaws are short is evident by the crowded condition of the teeth, even though but three premolars are present in each series (Fig. 24). The mandibular series is posterior to the normal relation with the maxillary series, and there is practically no effective occlusion. In the anterior regions the bite is open with the mandibular canines and incisors in a near horizontal position as in the English Bulldog. The protruding tongue is undoubtedly responsible for the open bite and the atypical inclination of the teeth.

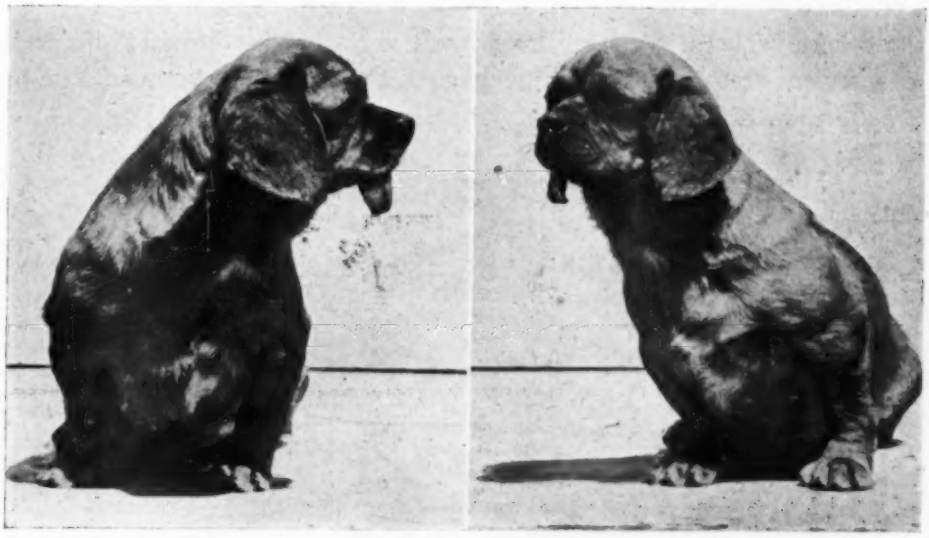

Fig. 23.—F_3 Dachshund by Pekingese.

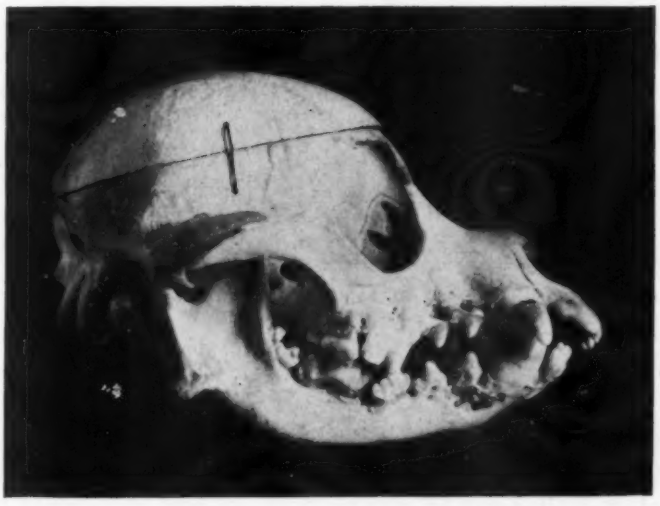

Fig. 24.—Skull of the animal in Fig. 23.

IV

The following experiments clarify to some extent the effect of modified environmental influences on skull form:

Impressions of the palates (Fig. 25) were taken at the age of two and one-half months and were made at frequent intervals for ten months. The top

row is the first of the series; the lower row, the last. From left to right, top row, at two and one-half months of age the pituitary gland was removed from Number 3; the thyroid from Number 4; the gonads from Number 5. The first model at the left was held as a control.

The lower row of five are models from impressions of the palates of the dogs of this litter at one year of age. The upper row does not include a model of the palate of Number 1 as we neglected to take an impression of it at two and one-half months. It is shown here as a control because it is a litter mate of the others. There is very little normal variation in the palatal size in the F_2 generation of this cross inasmuch as little difference between the palates of the German Shepherd and the Bassethound is apparent.

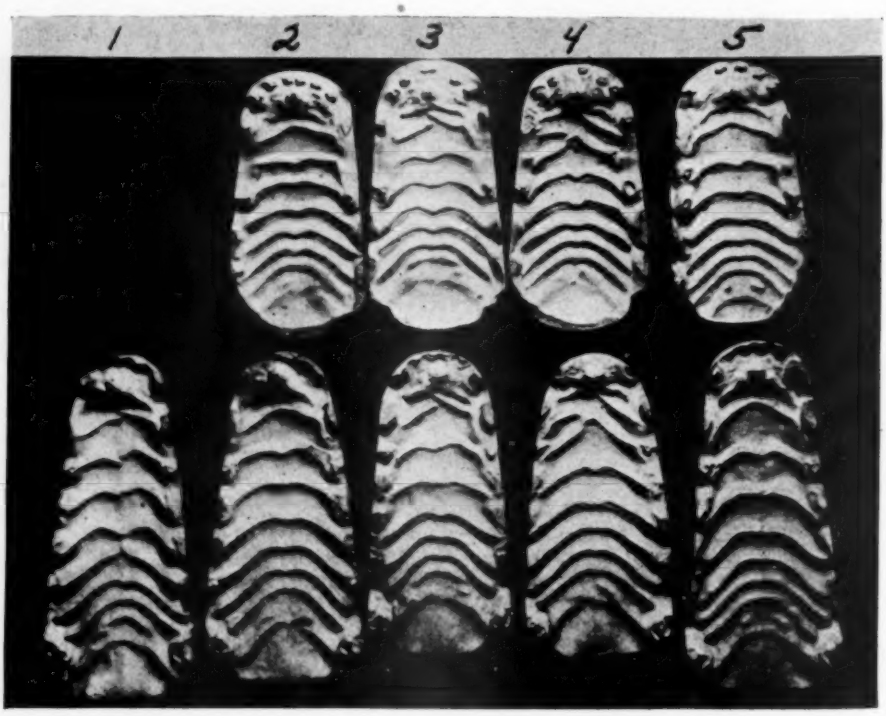

Fig. 25.—Models from impressions of palates of Bassethound by German Shepherd F_2 litter mates.

It is important to note that the palates of the hypophysectomized and thyroidectomized dogs after ten months are equally stunted in growth. Lack of growth in the anteroposterior dimension did not result in crowding of the premolars or change of occlusion because of the normal spacing of these teeth. If the premolars had been in approximal contact originally, of course they would have been crowded. In animals operated on at this age tooth form is not modified in the least.

Reduction in the anteroposterior dimension is evident in the increased palatal index and the lower upper facial index in Fig. 26. Higher percentages of the sums of the anteroposterior dimensions of the maxillary and mandibular premolars to the premolar regions of the maxilla and mandible indicate the stunting effect of the hypophysectomy on the growth of the jaws. Whether the

character of tooth structure was modified in these experiments is not known since no histologic studies were made. The operation had no apparent influence on the size of the teeth.

For one year several dogs were kept on a synthetic diet (Fig. 27), based on Karr's formula with slight modifications.* On this diet exclusively the animals were in perfect health.

At two months of age a Bassethound-Dachshund F_2 was given this diet minus two-thirds of the bone ash. No particular reaction to the calcium deficiency was noted until death occurred suddenly at the age of seven months seven days.

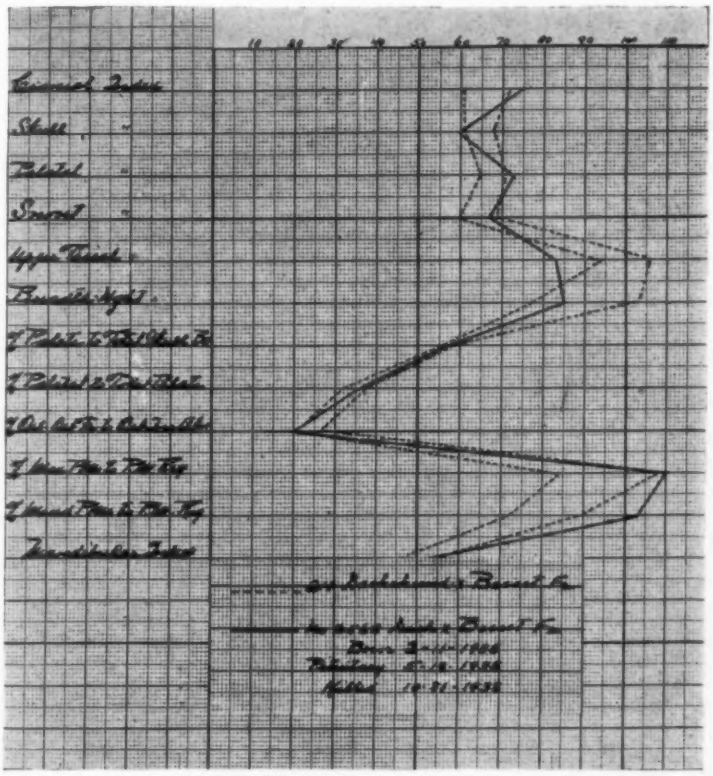

Fig. 26.—The effect of removal of the pituitary at three months on skull form as a whole on an animal from another litter of Bassethound-Dachshund stock.

In Fig. 27 the skull form of this animal is compared in detail with eighteen others of like genetic constitution on normal diet. There is no significant change. Although the skull was lighter in weight, its form was not modified.

From two F_2 Bassethound by Dachshund the bodies of the sphenoids with the osteogenetic cartilages, shown in Fig. 13, were surgically removed at the

*Karr's formula is found in Hawk and Bergeim, *Practical Physiological Chemistry*, 10th edition published in 1931 by Blakiston, pp. 694, 695.

Formula used: Percentages of salts: Na Cl 52%
Ca Lactate 22%
Mg Citrate 21%
Fe Citrate 5%
Iodine—a few drops to every 500 gms.

Other constituents:
Casein 37.6%
Sugar 34.9%
Lard 24.0%
Bone Ash 2.3%
Salt Mixture (above) 1.2%

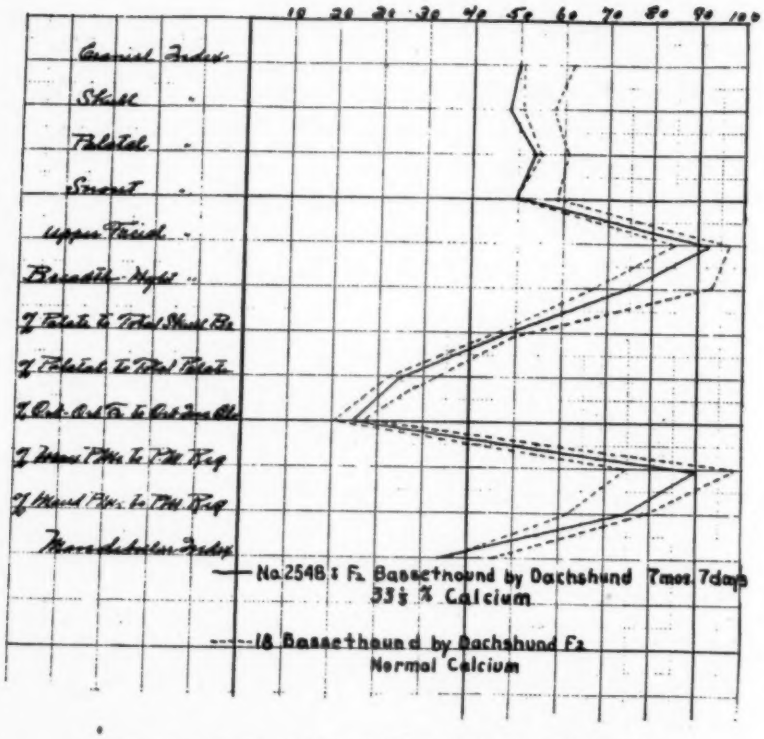

Fig. 27.—Effect of low calcium diet on skull form.

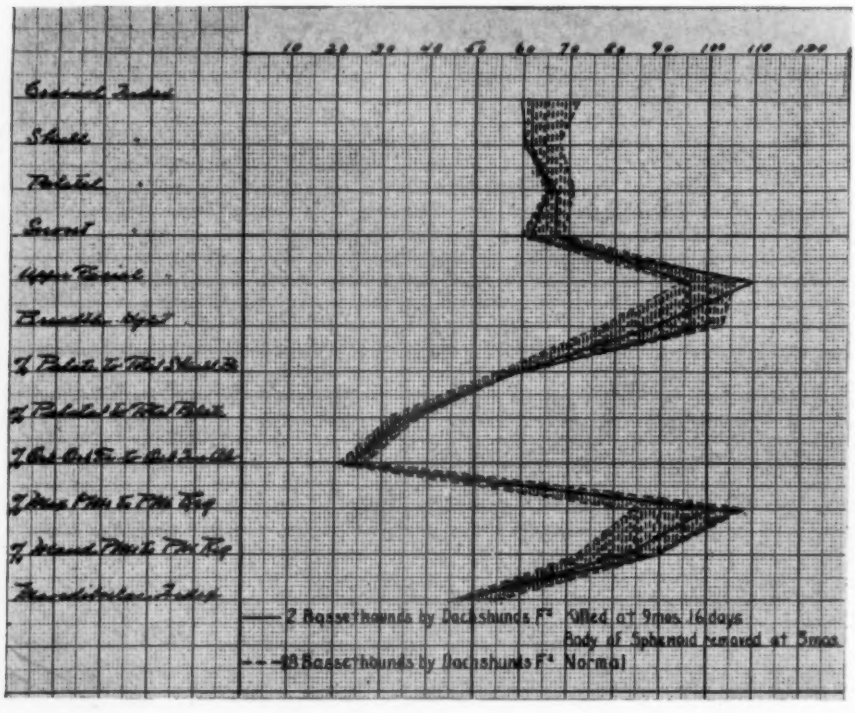

Fig. 28.—Surgical interference.

age of three months with the drill of the dental engine (Figs. 28 and 29). That there was little or no effect is shown in Fig. 28. Fig. 29 shows how complete was the regeneration of the parts removed. Except for the indistinct delineation of the suture lines in the specimen at the right, there is no evidence whatever of the mutilation. Dental occlusion was normal in both skulls.

V

The experiments in hybridization here reported demonstrate the fact that genetic constitution is a vital factor in the development of skull form and dental occlusion. The trend of the evidence presented follows very closely the Mendelian principles of inheritance. In each case the F_1 generation shows in skull form a decided similarity to the long-muzzled, normal ancestral type. The long-muzzled type is dominant over the short-muzzled, mutant form. As would be expected, great diversity in form is found in the F_2 generation. This generation ranges from a type comparable in most details with that of the long-muzzled, pure bred grandparent, toward the recessive modified type, though never duplicating it in the same degree as those at the other end of the series approach their long-muzzled forebears.

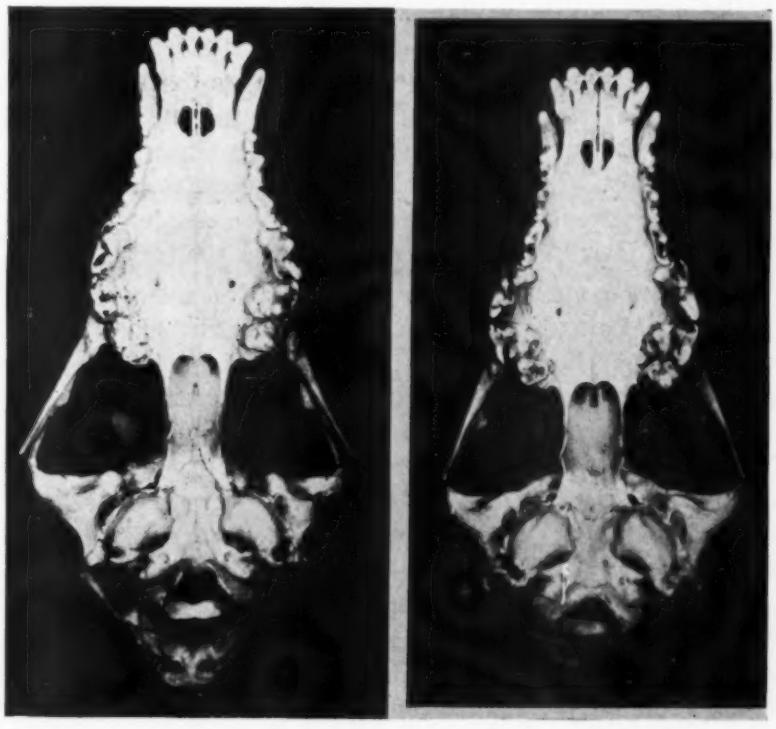

Fig. 29.—Surgical interference.

In each of the crosses reported one of the pure breds is so atypical that character peculiarities can be readily identified in succeeding generations. In passing from one generation to the next they follow Mendel's law of "the recombination of unmodified units" as they disappear and appear in different generations. Take, for example, the defective dentition of the Boston Terrier. Undoubtedly genetic factors play a very important role in the development of

this condition because of its consistent appearance in Boston Terrier with Boston Terrier mating under any living conditions where the animals can survive. The defective dentition of the Boston Terrier crossed with a Dachshund does not appear in the F_1 generation; it is evident in the F_2 generation. In other words, the mutant genes responsible for this defective dentition have come through the F_1 generation unmodified and appear in the F_2 generation in accordance with Mendel's law.

The independence in genetic constitution of the maxillary structures from the mandible and the independence of the teeth from both is demonstrated so clearly in the evidence that further comment is unnecessary. The implications of this truth, however, may warrant a brief discussion.

Tooth form is unquestionably the most stable character of the dog skull. But among pure breeds there is a certain correlation between the size of the teeth and the size of the dog. The teeth of a German Shepherd are larger than those of a Pomeranian. Hence crossing two breeds of the long-muzzled type which are widely different in size will produce a certain number of offspring with teeth too large or too small for the jaws. But the animals used in the experiments reported at this time, although not of exactly the same size, are so nearly alike that there is no significant difference in tooth dimensions. Yet they do exhibit a wide difference in tooth relations. Those with the short muzzle are crowded; those with the long muzzle are normal in alignment and occlusion. Because of the uniformity of tooth form and size the dental malocclusion in the specimens shown is the result of structural modifications of the osseous tissues supporting the teeth. These modifications are primarily an expression of genetic factors; and these genetic factors responsible for the atypical development of the jaws have little if any effect on the teeth.

A review of our material reveals the strong influence of germinal factors on skull form. It also shows that the skull is constituted of many characters, some dominant in inheritance, some recessive, and in the transmission of these characters from one generation to the next different combinations of the genetic elements responsible for them result in a variety of skull forms. Yet a certain degree of harmony in this mosaic of genetically more or less independent elements is obtained by the integrating factors of the internal environment of the organism. The point of emphasis here is that our experiments show that although the harmonizing influence of the internal environment is recognized as paramount in organization and development, it is not always sufficiently effective to reduce disharmonies of genetic origin. Crossing contrasted dog types demonstrates this truth: that structural disharmony, even to the extent of grotesqueness, may be an expression of the very nature of the organism itself. The assumption so often expressed in orthodontic literature that the hereditary tendency of all organisms is to develop the entire skull in harmonious design typical for the species, and that all departures from the typical are solely the result of environmental influences, is without scientific justification. That there may be a natural disharmony between the amount of tooth material and their supporting structures is a fact substantiated by the evidence here presented.

Undoubtedly the open-bite and horizontal inclination of the incisors and canines in Fig. 24 are due to the direct influence of the tongue. Many ortho-

dontists today are content with such mechanical explanations of most occlusal anomalies. But this case demonstrates the superficiality of such an attitude. The pernicious influence of the tongue arises from the disproportion in size between it and the bony structures of the face. This disproportion, being an expression of the genetic constitution of the animal, conditions all environmental influences which would tend to produce harmonious structural relations. From this clinicians can draw a suggestion of practical importance: that behind many so-called "tongue habit" cases there may be more basic developmental factors to be reckoned with in treatment; and in the degree that the presence and nature of these factors are understood will the service rendered be beneficial to their patients.

As the development of the skull is the result of the interaction of germinal constitution with environmental forces, the influence of the former was also studied through the modification of the latter. One type of experiment was performed to change the internal environment, i.e., the removal of certain endocrine glands. As a result of these operations skull form was not changed to any appreciable extent although there was evidence of a general arrest of growth or change in size. An interesting point to note, however, is that surgical removal of the endocrines of our dogs did not produce developmental duplicates of nature's experiments of endocrine imbalance in the human being. Obviously, abnormal growth expressions occur when the integrative action of endocrine function is surgically interfered with. But rarely if ever is it possible to obtain the classic pictures so often referred to in orthodontic literature. Moreover, breed differences condition the reaction of individuals to endocrine experimentation.

Another point of importance is that the endocrinopaths seen in such breeds as the St. Bernard, Bloodhound, and English Bulldog show in hybridization a definite genetic linkage. Certainly our knowledge of the specific parts played by the different endocrines in the development of skull distortions is very limited. Howard has called attention to the peculiar facial conformation of human beings with endocrine imbalance. He has made special reference to the so-called Class III condition which characterizes the acromegalic, suggesting that degrees of this condition which he aptly terms "acromegaloid," may be met with in orthodontic practice.[15] Nevertheless, most deformities are interpreted as developmental arrests of some part with adjustments of related structures to it. Our investigations suggest the probability that a larger number of the Class III cases are due to developmental arrests of the upper face with modification in the form of the mandible because of its adaptation to the distorted upper face. And these development arrests are, in some instances at least, an expression of genetic constitution.

Low calcium diet produced no appreciable difference in the form and proportions of the Bassethound-Dachshund F_2 skull even though it was started at three months, during the growth period and before the adult dentition was established. Evidently the reservoir of calcium in its own bones was sufficient to meet for a time the dietary deficiency. When an adequate supply of calcium was no longer available, the animal died in tetanus. Physiologic organization was maintained as long as possible by drawing on its own resources.

This experiment is reported here not in any sense as the final answer to the effect of diet on skull form, but to show the strong influence of constitutional forces in the face of an external environmental deficiency.

Surgical removal of the osteogenetic cartilages and the body of the sphenoid at the base of the skull was to determine the influence of their destruction on the growth of the face. In view of the theory so often expressed that these parts are largely responsible for the anteroposterior growth dimension of the skull base, it was at least a reasonable assumption that their removal might modify growth in this direction. That it had no marked effect is probably due to the relatively small amount of the chondrocranium removed. It seems that perhaps too extensive a growth influence has been attributed to these cartilages. In achondroplasia the entire cartilage of the skull is affected. Surgically we could not create a comparable condition. The skulls were constitutionally normal and the influence of the constitutional factors was strong enough to compensate for the elements destroyed by the operation.

VI

It is true that in skull types and dental occlusion the human being is quite different from the dog. The application of the results of animal experimentation to human requires bridging a gap which to some orthodontists raises grave doubts. Be this as it may, the principles of genetics, so far as we know them today, apply to all living organisms. The nature of chromosomal distribution, the appearance of dominants and recessives, and the problem of mutant factors have been studied in plants and animals of various kinds, and they reveal the same basic phenomena. Man is no exception. Moreover, until proof to the contrary can be adduced, it is safe to assume that the biologic relationship of the teeth to their supporting structures is much the same in man as in the dog.

In the light of the evidence here presented on the relation of germinal constitution to skull form there is room for little doubt that the primary factors of dental malocclusion in the dog are in the main an inherited condition. It is, however, a far cry to designate, on the basis of this investigation, just which types of dental malocclusion in human beings are inherited and which are not.

Many who venture into the mysteries of genetics are disappointed that concrete answers to practical problems are not immediately found. Such an attitude is unreasonable. In the exploration of a field which has been so consistently ignored as genetics, concentration on a single objective of assumed practical importance is inevitably shortsighted, for it limits vision to such an extent that essential factors likely to appear in most unexpected places are often overlooked. Before answers to specific questions can be obtained a general exploration, following any lead that may throw light upon the developmental phenomena of the face, is of primary importance. We must know, e.g., whether the "distoclusion" condition is ever present in the dog and whether it is a genetic character before we can state positively whether it is a dominant or recessive in inheritance. Genetics is a stimulating and profitable field for those interested in the etiology of malocclusion of the teeth. Nevetheless, a great deal of groundwork is necessary before trustworthy answers to clinical problems can be attained.

In this discussion I have endeavored to emphasize the necessity of including germinal constitution in the study of skull form and dental occlusion. On the basis of our philosophy there is no doubt of the belief that environmental forces influence the expression of germinal factors. Our insistence on control of living conditions and the study of the endocrines and nervous reactions of all the animals reported in order to identify conditions of genetic origin is evidence of this. On the other hand the material presented demonstrates a fact of great clinical significance: Germinal constitution conditions environmental effects. *Whatever extraneous influences are brought to bear on the whole or any part of the living organism in the form of physical, chemical, or social living conditions, even of an orthodontic appliance, the effect is determined to a variable but certain extent by the genetic constitution of the tissues.* In no way can the concept of individuality, which plays an important role in all our considerations, be better understood than by the study of genetics.

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A CINEFLUOROGRAPHIC STUDY OF THE HUMAN MASTICATORY APPARATUS IN FUNCTION

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THE great difference between orthodontics as it was understood and practiced some forty years ago and modern orthodontics is pointed out by Weinberger,[1] as follows: "It was in 1900 that Dr. Edward H. Angle defined orthodontia as 'that science which has for its object the correction of malocclusion of the teeth.' By 1927, the scope of the field embraced and the problems confronting the orthodontist had so changed that McCoy proposed the following: 'Orthodontia is a study of dental and oral development; it seeks to determine the factors which control growth processes to the end that a normal functional and anatomical relationship of these parts may be realized, and aims to learn the influences necessary to maintain such conditions when once established.'"

Our paper and the accompanying motion picture are of interest to the modern orthodontist because they deal with the process of mastication. Masticatory function is the main factor determining dental and oral development. It controls growth processes and, when properly used, helps maintain the teeth and arches in a normal, healthy state. On the other hand, abuse or disuse of the masticatory apparatus is one of the most important, if not the most important, etiological factors contributing to abnormal growth of the jaws and teeth and influencing their improper anatomic and functional development.

We are going to present here a cinefluorographic view of the human masticatory apparatus in function. Cinefluorography, as defined by Stewart,[2] "is the making of a motion picture record of the image seen on the fluoroscopic screen." Cinefluorography affords us an opportunity to see the internal phenomena taking place in the head and neck during the processes of mastication and deglutition, and enables us to study the organs which function in these processes.

On a previous occasion,[3] we presented a detailed description of the technique of cinefluorography. This essay, therefore, will only deal with the physiology of mastication and its importance to the orthodontist.

PHYSIOLOGY OF MASTICATION

When we speak of the masticatory apparatus, we mean all the organs and tissues which enter into the make-up of the complex and complicated machine that chews, crushes, grinds, and comminutes the food, making it suitable for swallowing and digestion. This includes the teeth, dental arches, muscles, tongue, glands, nasal passages, throat, and accessory sinuses and their blood, lymph, and nerve supplies. The process of deglutition is so closely interwoven with mastication that it is practically impossible to separate the two. Naturally, we cannot cover so broad a subject in this brief paper. We shall limit ourselves to the more salient features of masticatory function.

Read before the American Association of Orthodontists, Chicago, May, 1940.

Mastication is the process of grinding food between the upper and lower teeth brought about by the movements of the mandible against the maxilla. The muscles directly responsible for these mandibular movements are the masseter, temporal, and external and internal pterygoids. The movements are made possible by the peculiar manner in which the mandible is attached to, and articulates with, the temporal bones of the head—the temporomandibular articulation. This is a hinge-gliding joint, formed by the condyle of the mandible and the mandibular fossa and articular tubercle of the temporal bone. Connecting the two is a loose envelope, the articular capsule. Between the two is a thin plate, the articular disk, or meniscus. The condyle swings hingelike upon the articular disk. The disk, together with the condyle, glides forward onto the articular tubercle and back again. This articulation permits extensive movements of the mandible. Thus, the mandible may be depressed and elevated, carried forward and backward, and also from side to side.

Let us examine these movements in more detail. The mouth is opened by the action of the external pterygoid muscle (assisted by the hyoid group), which drags the disk and condyle forward on the articular tubercle, thus depressing the body of the jaw. The coronoid process is moved forward and downward, and the angle of the jaw moves backward and downward. The forward and downward movement of the coronoid process causes the elongation of the temporal muscle, while the backward and downward movement of the angle of the jaw stretches the masseter and internal pterygoid. In closing the mouth, these three great muscles press the lower teeth against the upper, and the condyle slips home to its socket.

When the upward movement takes place, we notice a double mechanism at work: the biting mechanism when the anterior teeth meet, and the grinding mechanism when the posteriors meet. When food is to be incised, the maxillary and mandibular incisors are brought into end-to-end relationship. This movement is carried out by the masseter and internal pterygoid muscles. When ordinary chewing movements are made, that is, when the lower molars grind against the uppers, the temporal muscle can be seen strongly at work.

When the mandible moves forward, the disk and the condyle glide forward on the mandibular fossa and articular tubercle. The movement is brought about by the internal and external pterygoids, the superficial fibers of the masseter, and the anterior fibers of the temporal.

In the backward movement of the mandible, the condyle slips back to its normal position in the socket. The muscles involved are the deep fibers of the masseter and the anterior fibers of the temporal.

In the side-to-side movement, one condyle with its disk glides alternately forward and backward, while the other condyle moves simultaneously in the opposite direction. This movement is brought about by the alternate action of the pterygoid muscles of either side.

The various movements are performed either simultaneously or in such rapid succession that it is impossible to distinguish one from the other during the process of mastication. Mastication, therefore, is a combination of all these dimensional movements.

Besides the above-named muscles, we must also note other organs which play a prominent part in mastication, namely, the tongue, cheeks, and lips. These are the muscular organs which control and direct the food onto the morsal surfaces of the teeth. The tongue also plays an important selective role in deciding whether particles of food are sufficiently small to be swallowed, and functions in the first stages of deglutition.

STRESSES OF MASTICATION

Until now, we have been considering the dimensional movements of the mandible. Now, let us examine the different degrees, extents, and intensities of these movements. These differ in every individual. They vary with the amount of force or stress capable of being exerted by the muscles of mastication. The amount of force, in turn, depends upon the extent of development, tonicity, and general health of the masticatory muscles. Quantitative measurements of the forces of mastication can be made.

The idea of measuring masticatory stresses is not a new one. As far back as 1681, an Italian scientist, Professor Giovanni Borelli, made a study of the strength of the muscles of mastication as compared with other muscles of the body. No other work in this field is recorded, however, until the end of the nineteenth century. At that time, interest in the subject was revived.

In 1895, Dr. G. V. Black[4] wrote on the gnathodynamometer and its importance in measuring the forces of mastication. More recently, Klaffenbach[5] used a gnathodynamometer to test the bites of one thousand students. After giving us the results of his experimentation, he comments: "It is interesting to note that Dr. Black found the average force exerted on the molar teeth to be 171 pounds, while we found it to be 150 pounds as recorded by the instrument, or 125 pounds actual force. Does this decrease of poundage in the force of the bite indicate that mastication pressure is gradually decreasing? If so, is this due to the fact that our foods now do not require the masticatory force formerly required?"

Let us compare these findings with those recorded for the Eskimo as reported by Dr. Waugh.[6] Waugh found the average for the Eskimo to be about 300 pounds, which is at least twice as great as that of the average American white person, despite the fact that the Eskimo is shorter in stature and shorter-lived than the white man. This can only be accounted for by the fact that the Eskimo develops this masticatory apparatus by use, while the white man suffers from lack of use, or disuse, of his chewing organs.

It has been pointed out by Black, Klaffenbach, and others that these measurements do not indicate the entire force of the muscles of mastication, since the person that bites on the gnathodynamometer usually stops as soon as he feels that it is hurting his teeth, the condition of the peridental membrane being the controlling factor. For this reason, your essayist some time ago undertook to investigate whether these measurements simply indicate the state of the peridental membrane or whether they also show the state of health of the muscles of mastication. We measured the bite of 100 young men and women between the ages of 13 and 23. The instrument used was the Haber gnathodynamometer. We recorded not only the maximum biting stress of each individual,

but also the amount of stress each individual used during ordinary mastication. A report of this work was published on a previous occasion.[7] We found that there was an approximate ratio of four to one between the maximum stress and the ordinary biting stress. For example: a person who was capable of registering 100 pounds maximum exertion at the molar teeth, recorded about 25 pounds in the same region during ordinary chewing. The one who registered 50 pounds maximum pressure, registered only 10 to 15 pounds during ordinary chewing. During normal masticatory function, when a person uses about a quarter of his maximum pressure, the condition of the peridental membrane certainly cannot be the controlling factor. Therefore, we can safely state that these measurements are indicative of the degree of development of the muscles of mastication.

Now, to go back to our subject, how can we explain the fact that the biting stress of the white man is so low as compared with that of the Eskimo? We find an answer to this question when we examine the difference in the type of food consumed by these two groups. This takes us to the very important subject of food and mastication.

THE EFFECT OF FOOD ON MASTICATION

Dental literature is replete with discussions of food and diet and their relation to dental and oral health. However, most researchers emphasize the metabolic and nutritional aspects of food. The vitamins and the inorganic salts hold the center of the stage, and food chemistry is more popular than ever. Such studies of diet and nutrition are as important to the dentist as any of the biologic sciences. But these are not special dental subjects. They belong rather to the domain of the pediatrician and general dietitian. The aspect of food and diet of especial importance to the dentist is the physical state of food and its mechanical significance in mastication. It is regrettable that so many overlook or ignore this subject.

Once the proper chemical factors are accounted for, the growth, development, and state of health of the organs associated with the masticatory apparatus are dependent on the degree of function of these organs. This, in turn, depends on the physical state of the food and the degree to which it stimulates mastication.

In making up a motion picture showing the process of human mastication, our main object was to demonstrate the difference in effect of stimulating and nonstimulating foods, and to show how the masticatory organs react to these foods. We see no action when liquid foods are taken, and very little movement when soft, nonfibrous foods are eaten. On the other hand, hard, bulky, fibrous foods stimulate the masticatory apparatus to great activity. We can observe the intense movements of the mandible and the strong action of the masticatory muscles. We note the strenuous action of the teeth in incising and then in chewing the coarse, resistant foods. With the aid of the fluoroscope, we can also observe the inner organs and parts of the masticatory apparatus. We see the dental arches and the teeth, and the bones of the head and neck. The swinging movements of the mandible are brought to view, as well as the changes taking place in the temporomandibular joint; the action of the tongue in tossing

about the food, directing it to the occlusal surfaces of the teeth, then to the pharynx for swallowing. And the very process of deglutition can easily be seen through the fluoroscope. There is a great deal of action and movement, a harmonious combination of living organs and tissues in a constant state of action.

We experimented with the following foods in our motion picture:

(a) *Milk.*—Chemically, milk is a very nourishing food. It contains proteins, carbohydrates, fats, minerals, and vitamins essential for the building-up of the body. But milk is a liquid, and does not stimulate mastication. It becomes the duty of the dentist to instruct the mother or pediatrician responsible for the feeding of the child that as soon as the teeth develop, the child should be given, in addition to milk, some solid food, such as a piece of orange or a crust of bread, in order to exercise his masticatory organs.

(b) *Soft Bread.*—This food is much eaten by the adult of today. Soft bread or cake, although a solid, stimulates very little mastication. To quote Dr. J. Sim Wallace[8] of England: "The food is simply taken into the mouth, receives a general squash between the teeth or between the dorsum of the tongue and the hard palate, and is then swallowed."

(c) *Orange.*—This is also a soft food, but the pulp of an orange is fibrous. Some chewing is required before it can be swallowed. The sensitive tongue will not permit the fibrous part to pass stomachward until it is properly comminuted. Therefore the pulp of an orange stimulates mastication. Here again is a case where the dentist can assert himself and say to the dietitian: "You claim that orange juice is good for the child because it contains the necessary vitamins. But why not give the child orange in its natural, solid form with the pulp, so that he receives its dental value as well as its nutritional value?"

(d) *Hard Bread.*—Hard bread is a great stimulant to mastication. When we chew a slice of bread, it is the coarse, resistant crust that causes hard, prolonged chewing. Therefore its use should be encouraged.

(e) *Celery.*—A vegetable such as celery provides much stimulation due to its marked fibrosity and hardness in the raw state. This effect is lost when the food is subjected to the softening influence of cooking.

(f) *Steak.*—Meat requires prolonged chewing before it can be swallowed, because it is fibrous. Steak not only requires *longer* chewing, but offers great resistance to the teeth and requires *more forceful* chewing. It is an excellent food as a masticatory stimulant.

(g) *Hard Corn.*—The degree of hardness of a food must not exceed a certain optimum state. When a food surpasses this optimum, mastication is retarded and interfered with. Therefore, hard, dry corn or raw rice is unsuitable for mastication and is not taken for food. We experimented with this substance because so many researchers used it in animal experimentation as the basis of their results. Some of the generalizations they present are quite illogical.

We tried these different foods on an individual with a full complement of teeth and well-developed masticatory apparatus. In the latter part of the movie, we contrasted this with the chewing process of an individual who lost

his posterior teeth. Then follows a view of an individual whose lost posterior teeth have been replaced by an artificial denture for the restoration of function. The radical changes in the process of mastication in these abnormal conditions are distinctly brought to our view. They can better be appreciated by a careful watching of the cinefluorographic part of the motion picture.

THE EFFECT OF MASTICATION ON GROWTH AND DEVELOPMENT

Proper function is essential to the health and development of all living parts of the body. The masticatory organs are no exception. If the muscles of mastication are subjected to the stimulus of a hard, bulky, resistant diet, they grow large and strong and develop a high state of tonicity. Lack of function, however, allows them to deteriorate and become soft and flabby.

We also know that bone is dominated by muscle. Its size and shape and structural strength vary with the degree of stimulation provided by the muscles which attach to it. A powerful, functioning masticatory musculature will thus lead to the development of prominent, well-built jaws and strong skull bones. In a similar fashion, the other tissues and organs of the masticatory apparatus are stimulated to full development by adequate function.

Primitive peoples, such as the Eskimo, subsist on an unrefined type of diet. The foods are hard, raw, and fibrous, and require long and powerful chewing. Their jaws, teeth, and muscles of mastication are strong, firm, and healthy. More civilized people resort to a softer, more refined type of diet which is easily swallowed after a minimum of mastication. Here we find that the jaws do not develop fully, teeth become crowded and impacted, and occlusion is distorted. Even the shape and appearance of the face are affected by the lack of function of the muscles of mastication. The celebrated anthropologist, Sir Arthur Keith, says:[9] "In the inhabitants of our Western cities the biting mechanism has fallen into disuse. The overlapping incisor bite has appeared. The cheeks, which are high and prominent when the biting muscles—the masseter and internal pterygoid—are well-developed, become reduced and sunken, giving us our narrow, hatchet-shaped faces—our oval cast of countenance."

Another anthropologist, E. A. Hooton, of Harvard, reminds us of the neglect of our bodies. He points out that civilization, with its wonderful inventions and labor-saving devices, is really doing us a great deal of harm. Our organs are allowed to remain functionless, and are not stimulated by use and exercise to maintain their health. This leads to their atrophy and degeneration. We wish to quote a few lines from Hooton as reported in the daily press: "Machines are getting better and better, while man gets worse and worse. The extraordinary tools are no longer accessories; the tail now wags the dog, and even thinks for him. It remains for the other end only to bark and bite." The only correction we wish to make is that, since modern man does so little "biting," there is nothing left for the other end to do but "bark."

While we do try to make up for the disuse of the other body muscles by artificial means, such as general exercise and calisthenics, the masticatory muscles do not even get this kind of treatment. Of all the muscles of our body, these are exercised the least.

Lack of function of the masticatory organs brought about by our civilized habits and refined methods of preparing food is the main factor responsible for the underdevelopment of the jaws and irregularities of the teeth. We realize that there are many other etiological factors which must not be overlooked by the orthodontist. But the factor of mastication should receive our greatest attention because it, more than anything else, contributes to the greatest number of abnormalities prevalent among our civilized communities.

CONCLUSION

Ideal orthodontics should emphasize the prevention of malocclusion. In its broadest sense, this means teaching the public how to develop and maintain a strong and healthy masticatory apparatus. Proper function, brought about by the use of stimulating foods, is the most natural way of preventing malocclusion. Those who plan children's diets should be instructed to include foods which encourage masticatory function, foods of the hard, bulky, and fibrous types. This may conflict with long-established habits and customs which are difficult to set aside. But if we wish to achieve practical results, we must fight and overcome the inertia which has always mired progress.

Dentistry is a health service, and as such is a branch of the medical science. The great advances in preventive medicine are due to the practical application of new ideas and discoveries to daily usage. Many times, this entailed changing radically the habits and customs of the people. The pages of medical history are replete with instances where the application of new preventive measures to check disease and promote health encountered enormous opposition from defenders of the status quo. It required organized effort on the part of the medical profession to overcome all these difficulties before new sanitary laws could be enacted and new habits, customs, and morals established. Let us emulate our medical confreres in our efforts for preventive orthodontics by advocating definite changes in the methods of choosing and preparing our foods. This will contribute to a healthier masticatory apparatus and improved facial esthetics.

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401 BROADWAY

THE INFLUENCE OF THE ENDOCRINES ON THE TEETH AND JAWS

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THE breadth of the subject, as assigned to this place on the program, is such that we would not have time to cover fully, or even partially, a comprehensive outline of all conditions of the jaws and teeth influenced by the endocrine glands. On the other hand, the title does not include the gingival tissues for discussion, which limits our field immensely. Probably then, a better and more inclusive title for this discussion would be "The Effect of the Endocrines on Dental Development."

In the preparation of this paper, as well as in association with members of your profession, I have been impressed by the different position occupied by the dentist of today as compared with that of his predecessor of only a few decades ago. We look upon the dentist today as a specialist in diseases of the oral cavity. The rapid advances which have been made both in medicine and dentistry have emphasized more and more that the various structures which compose the oral cavity are part and parcel of the organism, constituting the portal of entry for food and air. The structures of the oral cavity prepare the food for, and initiate, the process of digestion, so that disease or disorder of the mouth may produce evidence of trouble in distant parts of the body. When we consider carefully how impossible it is to separate and treat one portion of the organism without due consideration of the organism as a whole, the truth of the following words from that ancient Grecian philosopher, Socrates, becomes apparent. He states: "I daresay you have heard eminent physicians say to a patient who comes to them with bad eyes, that they cannot treat the eyes by themselves, but that if the eyes are to be cured, the head must be treated. And then again they say that to think of the head alone, and not the rest of the body also, is the height of folly. And arguing thus, they apply their methods to the whole body, and try and treat and heal the whole and the part together." If we substitute "teeth" for the word "eyes," we should have expressed the aim of every true oral specialist, who is keenly aware of the fact that there are many internal diseases which exhibit manifestations most readily observable as alterations of the oral structures. With the increasing awareness of the public in regard to its health, it is becoming more incumbent upon the professions of medicine and dentistry to be in a position to prevent disease rather than to be content to attempt to cure after the disease has become evident. The dentist today occupies a position of ever-increasing importance in relation to preventive medicine.

The body politic consults its dentist more regularly for periodic check-up than it does its physician. This may be due to greater activity along this line by the dentist, but another and more potent factor, I believe, is that loss of the teeth or disease in the oral cavity impresses itself more concretely upon the individ-

Read before the meeting of the Southwestern Society of Orthodontists, February, 1940, Houston, Texas.

ual's attention than does incipient disease of other parts of the body. The establishment of prenatal and preschool clinics, in which dentists have their rightful position, results in children being brought under the care of the dentist at a much earlier age than formerly. It is not, I believe, uncommon for children of four to six years of age to be regularly seen by their dentists, whereas formerly it was extremely uncommon for a child to be taken to the dentist before the age of 10 or 12 years. This added responsibility the dentist has met by extending his investigations to the structural and functional status of those parts of the organism which to him, as an oral specialist, have been brought under suspicion. With such knowledge, correctly applied, the treatment of many baffling conditions becomes more effective, and many somatic ailments can be discovered before irreparable damage has made prognosis for life or cure hopeless.

The nutrition, growth, and repair of all tissues of the body, including those of the oral cavity, are dependent upon normal metabolic functions, and they in turn depend upon normal and correlated activities of the endocrine glands. Since the eruption, and later the maintenance of the teeth, and the integrity of the surrounding tissues are largely under the control of the endocrine glands, it is of importance to be ever mindful of the particular change in the mouth which may result from certain endocrinopathies.

No subject of recent years has excited quite the interest or produced so many fantastic theories as has the study of endocrinology. Some of the earlier books on the endocrines read like fantastic fiction and have a Wellsian flavor in their weird deductions. For almost every deviation from normal in function or structure, a specific endocrine gland was incriminated, and in like manner specific medication of the supposed active principle of this gland was advocated as a cure for the condition. This early confusion and misconception is understandable now, when we consider the known peculiar and widespread effect of the individual endocrine glands. These glands are singular in that they are adapted to secrete individual hormones at suitable times and in graded quantities, and their effects are so widespread that no organ or even part of an organ can escape their dominating influence. This is a complicating factor in the diagnosis and treatment of endocrine conditions, since the affected parts invariably produce secondary and tertiary effects in other organs, which in their turn affect the functions of a new set of endocrine glands with still further disturbances throughout the body. More confusing even than the above is the consideration of the degree of response or acceptability of the affected organ or structure reckoned with. The recent studies by Sure and Theis, and their coworkers at the University of Arkansas, should be a further stimulant to conservatism in considering the effect of endocrines on the organism. Their experiments have demonstrated the influence of avitaminosis on the endocrine glands as well as the effect of certain endocrine diseases, as thyrotoxicosis, on the vitamin content of the tissues. This condition, they have found, causes a depletion of vitamins B and C, with a resulting secondary effect upon the organism and the endocrine glands, due to this deficiency. It is not surprising, therefore, that the enthusiasm of some writers on endocrinology apparently gets the better of their judgment, and all too much importance is placed on the endocrine influence on

the organism without due regard to other conditions. In no other subject is it so necessary that a sane and critical attitude be maintained if we are to keep our feet on solid ground. It may be well for us to remember the remark credited to the humorist-philosopher Artemus Ward: "It is not so much that we are ignorant, but we know so much that is not true."

In an excellent article appearing in the *Journal of the American Dental Association* in 1934, Schour states: "Unwarranted or exaggerated claims have been reported on the effect of the endocrines on the teeth as a result of misconception and insufficient appreciation of some elementary fundamentals about the teeth. The two chief misconceptions are (1) that teeth are subject to change throughout life as are other tissues or organs, (2) that dental tissues undergo an early period of development, after which they do not change. The truth (he states) apparently rests between these two theories." Schour continues: "The first conception is incorrect because certain characteristics of the teeth, such as the shape and size of the crown, once determined by the enamel organ, are permanent and unchangeable, except by trauma or surgery. Enamel, once formed completely and calcified, is not generally believed to become modifiable except by exogenous means. The second conception is incorrect because the investing dental tissues, the gingivae, the periodontal membrane, the cementum, and the alveolar bone can change readily even long after the tooth has come into occlusion. There is increasing evidence that dentine too may be subject to modification in the teeth of the adult."

Since the size, shape, and integrity of all teeth are determined before they erupt, these factors can be influenced by endocrine disturbances only if they are operative at a time when the teeth are developing and are still submerged. For example, a calcium deficiency occurring during intrauterine life or shortly thereafter, will affect the calcification of the deciduous teeth while the permanent teeth will show the effect of such a change in metabolism if it occurs before the age of five or six years. Changes after this age, will only be reflected in the alveolar tissues. For any endocrinopathy to produce changes in the oral structure it must occur when the function determining the anatomic integrity is in the kinetic stage.

For convenience it may be well to review briefly the various glands of the endocrine system, and to consider their functions under normal conditions, and then to consider the controlling effect of the glands upon growth during fetal life and infancy and the mechanism at puberty. It is at these periods of life that changes in the oral structures occur, and the influence of the endocrines on such changes is our consideration at this time. Consideration of the functions of the glands of the endocrine system under normal conditions will help us to appreciate more clearly the changes produced by abnormal function.

The pineal gland, located near the center and posterior part of the cerebrum, is still a more or less undiscovered field. Adenomatous tumors cause rapid and excessive growth in the child, precocious sexual maturity and mental development. Death usually results early because of the pressure effect of the tumor, though advances in neurosurgery now hold out some hope for cure. No effect

on the teeth is noted except, of course, premature appearance accompanying the precocious development in other parts of the body.

The pituitary gland, located in a fossa, the sella turcica, at the base of the skull, is attached to the under and anterior part of the cerebrum. It consists of two distinct parts with totally different functions. The posterior lobe is derived from brain tissue and elaborates several hormones, which in association with certain nerve centers in the base of the brain, act to control water balance, body temperature, and part of carbohydrate metabolism. No effect upon the teeth has been noted either by administration of large doses of posterior pituitary extract, or by surgical removal of the posterior lobe in experimental animals.

The anterior lobe of the pituitary is glandular in structure and is a potent factor in body development. The growth hormone, which this gland elaborates, controls the growth of all tissues. Excess production of this hormone in the prepubertal era, before ossification is complete, results in gigantism of a symmetrical type. Damage severe enough to prevent the formation of this hormone results in cessation of growth and later the premature appearance of the degenerative changes of old age. If there is an excess production of the growth hormone after puberty, overgrowth of an asymmetrical character occurs, clinically represented by cases of acromegaly.

As puberty begins, the anterior lobe of the pituitary elaborates the sex hormone which controls the development of the external and internal genitalia. The growth hormone is also usually increased at this time and is followed later by a definite decrease. Marked alteration in the normal development of the jaws and teeth may result from disturbances in the production of the growth hormone. Only brief mention may be made of some of the many other hormones elaborated by the anterior pituitary lobe. The thyrotropic hormone stimulates thyroid activity, the adrenotropic hormone the cortex of the adrenals, and the parathyrotropic hormone the parathyroid glands. A galactagogue hormone has also been described as well as the bromine hormone, which seems to be connected (at least in dogs) with sleep. Zondek says that instead of so many different hormones arising from so small a gland, it may be assumed that these various substances are but modifications derived from a chemically uniform basic substance, by comparatively insignificant molecular changes.

The thyroid gland lies just below the larynx, and enlargement of this gland is known as goiter. In fetal life and early childhood the differentiation and maturing of tissues is controlled by this gland, while in later life by the regulation of the rate of oxygen consumption and energy production in each body cell, it controls the rate of function of tissue. Dental problems are greatly influenced by this function.

The parathyroid glands, usually four to six in number, are located posterior to, or in, the thyroid gland. Their function is principally concerned with the control of calcium metabolism. Complete removal or severe damage to the parathyroids results in a loss of the ability to mobilize calcium, with resulting hypocalcemia as low as 4 mg. per 100 c.c. of blood, and a clinical condition known as tetany. This is characterized by an extreme hyperexcitability of the

entire nervous system and frequent spasms, usually limited to the four extremities. Enamel hypoplasia has been noted in children who have suffered from tetany. This may show as a single or parallel rows of bands superposed one above the other, horizontally across the teeth.

Tumors of the parathyroids result in excessive activity of the glands, with a resultant abnormal mobilization of calcium from the depots in the body. The blood usually contains a high percentage of calcium, and there is loss of large quantities of calcium through the kidneys and bowels. Withdrawal of calcium from the bones results in cystic changes, or a general softening of the entire bony structure. Unfortunately, there is no evidence to prove that enamel once formed is subject to withdrawal, so that an apparently logical explanation of dental caries cannot be entertained. A distinct disease of the enamel may occur in hyperparathyroidism. A. Hess and his co-workers have described these poorly formed teeth in poorly formed alveolar structures as "enamel disease." A bluish color has also been noted in the teeth of the overactive parathyroid case.

The thymus gland located in the anterior part of the chest, above the heart, interests us because of the body changes occurring with thymic hyperplasia. Children so affected have short, thick necks, square chests, short extremities, and are extremely susceptible to trauma and infection, death frequently resulting from some trivial incident. No effect upon development of the teeth or jaws has been noted in the thymic child, though a palatal arch, high or irregular in shape, has been said to occur as a result of thymic disturbances, and points to incomplete descent of the nasofrontal process.

The suprarenal glands lie one above each kidney and consist of two portions, the medulla and the cortex. The medulla produces the hormone adrenin, which acts to increase the tone of the sympathetic nervous system at its nerve endings. The suprarenal cortex is the most essential portion of the gland. A substance called cortin is elaborated, which is essential for life, and ascorbic acid or Vitamin C has been found stored in this portion of the gland. Sodium, potassium, and nitrogen excretion are also controlled by the cortex. Along with the anterior lobe of the pituitary, the cortex of the suprarenal gland cooperates in the development of the sex organs at puberty, and also in their normal function after puberty. Tumors, resulting in hyperfunction, produce in children a sexual precocity and premature maturation of the sex organs. No particular characteristic changes have been noted in the development of the jaws or teeth in suprarenal conditions, though stress should be laid on the appearance of brown pigmented spots in the mucous membranes of the mouth and gum tissues, occurring in the hypofunctioning state of the cortex known as Addison's Disease.

The sex glands or gonads begin to mature with the onset of puberty, and are associated with the development of the secondary sex characteristics, adult type of hair, breasts, and general distribution of fat. The gonadal hormone has an inhibiting influence on the growth hormone from the anterior pituitary gland, so that if a deficiency of the gonadal hormone occurs, growth continues beyond the normal time. A resulting disproportion between the growth hor-

mone and the inhibiting gonadal hormone may involve the jaws, if the error occurs at the appropriate time. Disturbances in gonadal activity, after growth is completed, have no such effect.

At this point I would like to show some charts taken from Wolf's *Endocrinology in Modern Practice.* The impression gained from a study of these charts is that a definite endocrine change is responsible for almost every oral condition. In comparison to these charts, a summary of the paper "Endocrines and the Teeth" by Schour, referred to above, is in sharp contrast. The pineal gland he dismisses with the brief statement that there is so little that has been definitely established concerning this body that it will not even be discussed. In referring to the thymus gland he states: "The data available on the function of the thymus and its effect on the teeth are conflicting and variable. Also in view of the fact that a recent authoritative review on the physiology of the thymus concludes that there is no evidence of a specific endocrine function of the thymus, it seems rather far-fetched to speak of 'thymodontia.'" His treatment of the effect of the other glands on the oral structures is comparably conservative, and a summary of his conclusions has been referred to earlier in this discussion.

SUMMARY

DISEASES OF THE ORAL CAVITY

THERE MAY BE:

In Hyperthyroidism

1. Occasional presence of one or two teeth at birth.
2. Frequent dental caries due to drainage of calcium from system.
3. Delicate, thin maxillary bones.
4. Increase in salivation.
5. Necessity of care in drilling and use of anesthetics.

In Hypothyroidism

1. Delay in dental development.
2. Large central incisors.
3. Small lateral incisors.
4. Overlapping teeth.
5. Ridging of teeth.
6. Congenital malformations of mouth and teeth.
7. Malocclusion.
8. Chronic infection of teeth, tonsils and sinuses.
9. Abnormally soft dentine and enamel.
10. Root absorption extreme.
11. Lack of density in crowns of teeth.
12. Decalcification of carpal bones and superior maxillae.
13. Thickening of lips and tongue due to mucoid infiltration.
14. Hypertrophied tonsils and lymph nodes.
15. Pyorrhea.
16. Poor circulation in gums.
17. Tendency to canker sores.
18. Dry mouth.

In Hyperpituitarism

1. Accelerated appearance of teeth.
2. Large, square teeth.
3. Broad upper incisors.
4. Errors of alignment resulting from widely spaced teeth.
5. Teeth resistant to infection and caries.
6. Markedly hypertrophied lower jaw.
7. Enlarged lips and tongue.
8. Density of jaw bone renders tooth extraction difficult.

In Hypopituitarism

1. Delay in appearance of teeth.
2. Small lower jaw, causing marked overbite.
3. Small infantile teeth.
4. Stunted canines.
5. Large, well-spaced upper central incisors.
6. Rapid decay, crowns of teeth lacking density.
7. Overcrowded teeth and various other types of orthodontal conditions.
8. Bluish tint of teeth.
9. Congenital malformations of mouth and jaws.
10. Possible connection with pyorrhea.

In Thymic Hypertrophy

1. Delayed appearance of teeth.
2. Small upper maxilla.
3. Large central incisors.
4. Stunted lateral incisors.

(From Wolf's: *Endocrinology in Modern Practice.* Copyright W. B. Saunders Company.)

Returning to the consideration of the controlling effect of the endocrines upon growth during the kinetic periods of life, the fetal period, infancy and puberty, a consideration of changes dependent upon thyroid dysfunction will be discussed. It is with decreased activity of the thyroid hormone that we are concerned, as no changes in the growing organism have been noted as a direct result of hyperthyroidism. Deficiency of thyroid activity is probably more prevalent than we realize. In a review of our office cases a few years ago, we found that about 35 per cent showed basal metabolic rates below 10 per cent. This agrees rather closely with the experience of Marinus in a study of more than 5,000 children, as he found approximately one-third of all cases observed showing evidence of thyroid deficiency. We find, as a result of a deficient thyroid secretion, the late appearance of the deciduous teeth, and that they are incompletely formed and poorly calcified. Caries may result early, and there is an early loss of the teeth from the jaws, the first molars particularly, with the

DISEASES OF THE ORAL CAVITY

DISEASES OF THE ORAL CAVITY—CONTINUED

5. Crescent-shaped grinding surfaces.
6. Extensive caries, poor enamel.
7. Congenital malformations of mouth and jaws.
8. Hypertrophied tonsils and lymph nodes.
9. Pyorrhea.
10. Necessity of care in use of anesthetics.

In Diabetes

1. Premature appearance of teeth.
2. Extensive caries.
3. Abscesses of roots.
4. Swollen edges of tongue.
5. Abscesses and fissures of tongue.
6. Extensive gingivitis and ulcerations.
7. Pyorrhea.
8. Stomatitis.
9. Dry mouth.
10. Increased cholesterol in saliva.
11. Fruity breath odor (acetone).
12. Burning metallic taste in mouth.

In Hyperadrenalism

1. Premature appearance of teeth.
2. Unusually large canines.

In Hypo-adrenalism

1. Strong teeth, resistant to infection.
2. Yellowish discoloration of teeth.
3. Angioneurotic edema of tongue.
4. Allergic gingivitis.
5. Discoloration of oral mucous membrane.

In Hypergonadism

1. Frequent dental caries.
2. Often loss of teeth before 20 years of age.

In Hypogonadism

1. Stunting of laterals and canines.
2. Missing laterals in some cases.
3. Dental caries in adolescence, transition period of menopause frequent.
4. Small maxilla.
5. Possible connection with pyorrhea.

In Pregnancy

1. Caries due to loss of calcium.
2. Discoloration of teeth.
3. Hypertrophies in the gums.
4. Gingivitis.
5. Paresthesias of oral mucosa.

In Hyperparathyroidism

1. Decalcification of dentine, rendering cavities frequent and cavity preparation difficult.
2. Root absorption.
3. Giant cell tumors of the jaw.

In Hypoparathyroidism

1. Atrophic, peg-shaped teeth.
2. Defective enamel covering.
3. Lateral erosion of teeth.
4. Bacterial invasion of teeth and sinuses.
5. Breaking of teeth.
6. Horizontal rifts, ridging and furrowing of enamel.
7. Congenitally small mouth.
8. Possible connection with pyorrhea.

In Pineal Tumors

1. Premature appearance of teeth.

(From Wolf's: *Endocrinology in Modern Practice.* Copyright W. B. Saunders Company.)

resultant deficient jaw development with which you are familiar. The permanent teeth are usually late in appearing, and are irregular in size and poorly developed. Caries results, probably because of the faulty development. The changes noted in the deciduous and permanent teeth cause disturbances in occlusion and jaw development which produce severe orthodontic problems.

The conditions produced by deficiencies of the pituitary gland are usually manifested in the development of the jaw, the teeth themselves not being affected directly. The changes usually become apparent about the age of puberty. There may be delay in the normal increase of growth, or instead of uniform growth we may find increased growth in certain areas with delayed growth in others. This may result in various disturbances in the development of the jaws, such as an almost complete failure of development of the mandible, with normal development of the maxilla. The retention of infantile facial proportions with the eruption of normal-sized teeth is not an uncommon finding of this condition, and as a result there is a marked crowding of the teeth with irregularity in position. Overdevelopment of the mandible, with prognathism, resulting in malocclusion, may occur. Facial asymmetry may also be seen as a result of a relative overdevelopment of either the right or left side.

An excess of the growth hormone may occur, and depending on the time of life that this occurs, various changes will be noted. Symmetrical overgrowth occurs if the change manifests itself before any of the bone structures have completed their development, and no anomalies of the oral structures are noted. If, however, this excess of the growth hormone occurs after certain of the bone structures have completed their development, irregular overgrowth results. If the bones of the head have not completed their development and this excess of the growth hormone occurs, we find a widening of the dental arches with separation of the teeth and a loss of mutual support.

The condition known clinically as acromegaly is usually the result of an excessive production of growth hormone by a pituitary tumor after the long bones have completed their growth. The effect on the face is the production of an extreme overgrowth of the mandible and wide spacing of the teeth, with severe malocclusion resulting.

Gonadal changes do not of themselves directly affect the oral structures, except as regards the gingival tissues. In men, deficiency of gonadal secretions has resulted in recession of the gum tissue and signs of alveolar atrophy. Pyorrheic conditions and ready bleeding of the gums have also been noted. Gingival changes have been observed in women at puberty, and just prior to menstruation. In pregnancy a high incidence of gingivitis occurs. Ziskin and Blackberg proved the endocrine origin of this type of gingivitis by producing experimentally hyperplasia of gingival epithelium, resembling pregnancy gingivitis, by injecting monkeys with sex hormones. The intimate relationship of the gonads to the thyroid and pituitary results in gonadal changes being manifested in the tissues as more distinctly of a thyroid or pituitary dysfunction. Ill-defined changes in the gingivae and peridental membranes may be seen as a result of combined deficiencies of the gonads, thyroid, and pituitary, usually as a multiglandular manifestation.

In diabetes, which may be considered as an endocrine disease because of the failure in function of the islands of Langerhans in the pancreas, frequent changes in the gums occur. Pyorrhea and gingivitis are common accompaniments of diabetes, and it behooves the dentist to be cognizant of the fact and to ascertain definitely whether this disease is responsible for such changes coming to his attention. Not infrequently the first intimation that a patient his diabetes, may be the result of a urine examination urged by an alert dentist.

After a discussion of various effects that the endocrine glands may have on dental development, the natural question to be raised is concerning treatment. As we have been considering more or less new ideas of function and growth, we should expect new and specific medication for the changes resulting. When such an occasion arises, it is well to remember the words of Alexander Pope, in his Essay on Criticism:

> "Be not the first by whom the new are tried,
> Nor yet the last to lay the old aside."

As the effect of any endocrine disturbance can only be modified during that period when there is growth of the structure, changes in tooth formation occurring as a result of thyroid deficiencies would have to be treated during the fetal or early infancy period to produce any results. In suspected cases, a fairly definite diagnosis of thyroid deficiency can be made in the new born infant, and medication started immediately. The obstetricians recognize the influence of the thyroid on a normal gestation, and it is a frequent practice now for them to administer thyroid to both the potential and proved cases of pregnancies. This undoubtedly has a beneficial influence on mildly subthyroid individuals, and results in better tooth structure for their offspring. The ever increasing activity of the pediatrician, with supervision of the child beginning during the first weeks of life, results in earlier recognition of endocrine changes in the child, and the institution of treatment at a time when not only having a beneficial effect upon his physical growth and mental development, but also early enough to definitely influence dental development. As children are now going to the dentist at a much earlier age than formerly, recognition by the dentist of the thyroid effect upon the deciduous teeth will enable him to urge treatment early enough to have some effect upon the permanent teeth. It is obvious that no internal medication can influence tooth formation after the tooth has developed.

At the time of puberty, growth changes dependent upon gonadal and pituitary deficiencies may occur, and when those changes which have been discussed are recognized by the dentist, considerable benefit may be expected by the proper medication, intelligently given. We must remember, however, that unfortunately most of the cases coming to the dentist for treatment have passed the period of tooth development, and only the orthodox orthodontic procedures can be applied. The use of expensive medication is unnecessary and extravagant in such endocrine conditions where the structures have already reached complete development. The application of tried and proved mechanical principles in the treatment of such orthodontic problems certainly offers a greater expectancy of successful results than does endocrine therapy at the

present time. The use of endocrines as an adjuvant in properly selected cases is advisable and helpful, and it is the informed orthodontist who will refer such patients for treatment at the hands of internist or pediatrician.

Having quoted freely from the writings of Marinus, Wolf, Schour, and others, to whom grateful recognition is made, I recall a statement chronicled by Frank Case in his amusing *Tales of a Wayward Inn;* he repeats a phrase of Wilson Mizner's, who says: "When you take stuff from one writer it's plagiarism—but when you take from many writers, it's called research"! I trust that my remarks will appear to you as being in the latter category!

We have learned long since that the art of healing is too extensive and exhaustive for one person to be a master of all branches, so that in caring for oral disease the close association and consultation of the obstetrician, pediatrician, internist, and orthodontist will be productive of the best results. Such care of patients will result in the fullest application of the advice of Socrates, quoted at the beginning of this paper, and paraphrased in conclusion: "I dare say you have heard eminent *orthodontists* say to a patient who comes to them with *bad teeth,* that they cannot treat the *teeth* by themselves, but that if the *teeth* are to be cured, the head must be treated. And then again they say that to think of the head alone and not of the rest of the body also, is the height of folly. And arguing thus, they apply their methods to the whole body, and try and treat and heal the whole and the part together!"

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Erratum

The article, "Bone Changes Resulting From Experimental Orthodontic Treatment," by Dr. Carl Breitner, which appeared on page 521 of the June issue of the JOURNAL, should have carried the footnote, "Read before the New York Society of Orthodontists, New York, N. Y., March 11, 1940."

REPORT OF A CASE OF A SUBMERGING FIRST MOLAR

D. S. Sterrett, D.D.S., Erie, Pa.

The following case history is reported by Dr. Sterrett in the hope that some of the readers of the American Journal of Orthodontics and Oral Surgery will feel inclined to comment on the case as a result of experience.—Editor.

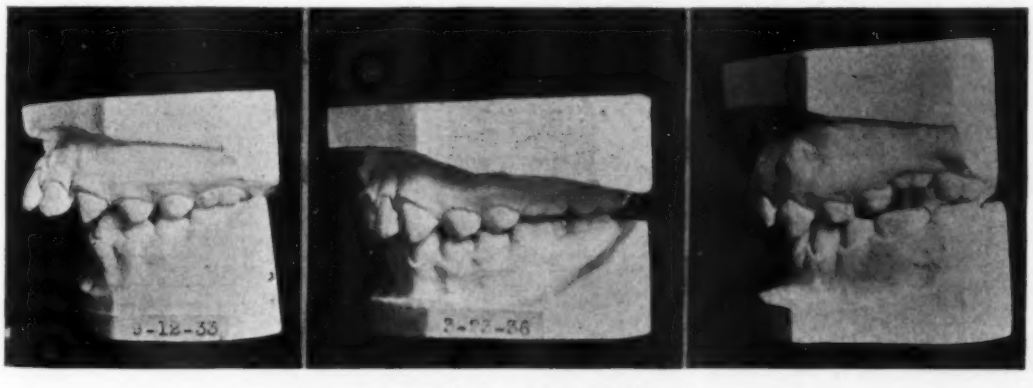
A GIRL, aged 9 years, 1 month, was placed under treatment in October, 1933, for the condition shown in profile cast, Fig. 1.

All deciduous teeth had been succeeded by the permanent teeth; there was an early thumb-sucking habit. The tonsils and adenoids were removed at the age of 5 years, with frequent infections and mouth breathing.

In June, 1932, she weighed 59¾ pounds and her height was 52½ inches. In September, 1933, she had gained to 76¾ pounds and height 55¾ inches, during a year of frequent illness, including pneumonia.

Mechanical treatment consisted essentially of a maxillary labial round wire in Angle bracket bands on the central incisors, canines, and molar sheaths, followed in five months with a ribbon arch, lower lingual arch, and intermaxillary rubbers.

The case progressed through 1934 despite some interruptions from illness, and in January, 1935, she had scarlet fever, followed by a chronic cough, but was well enough by March, 1935, to resume treatment. The patient was sick most of the following September, with afternoon temperature. She was seen once in October, but missed the next appointment because of a cold. She was then seen three times before another interruption from a cold in January, 1936.



Figs. 1 to 3.—Profile casts.

In March, 1936, all appliances were removed, and study models were made to determine, in view of past difficulties in carrying on treatment, whether to try more treatment, or retain, or entirely discontinue. At this time a depression or submerging of the maxillary left first molar was noticed, amounting to 2 mm. of clearance from the mandibular tooth (Fig. 2).

The girl's weight and height in March, 1936, were 112¾ pounds and 63¼ inches, respectively, and by July, 118 pounds and 63¾ inches. A Hawley retainer was placed on the maxillary teeth to try to hold the tooth positions in a fair pattern. In November, 1936, her weight was 127 pounds, and her height was 64½ inches; in January, 1937, 134 pounds and 65¼ inches.

The first molar seemed to be getting shorter and arrangements were made for study models and x-ray pictures for March, 1937. This was prevented by sickness and various delays so that it was not until February, 1940, that they were made. The profile cast, Fig. 3, taken Feb. 13, 1940, shows the maxillary left first molar 4 mm. above the mandibular molar.

Pictures of the x-ray films of that date seemed to show that the left antrum dipped deeply toward the interproximal space between the second premolar and first molar. There was no proximal contact between the left canine, first and second premolars, and first molar; so it seemed unlikely that the third molar was an important factor.

The question arises as to whether the posterior wall of the antrum, lying close to the first molar roots, is affecting the position of that tooth, and whether anything is growing in the antrum to affect its shape and size. Pictures of x-ray films of 1932 are included to show the only x-ray evidence when the child was first seen.

Fig. 4. Fig. 5.

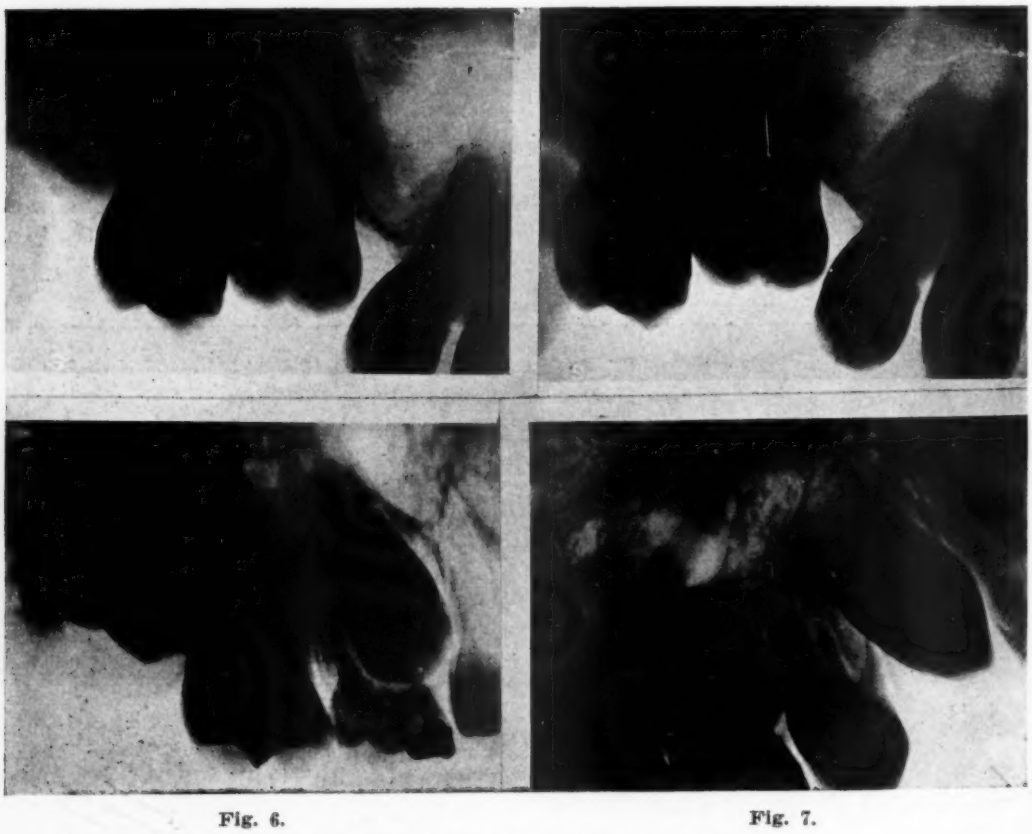


Fig. 6. Fig. 7.

Fig. 4.—X-ray of maxillary left molar region, February, 1940.
Fig. 5.—X-ray of maxillary left molar region, February, 1940.
Fig. 6.—X-ray of maxillary left molar region, July, 1932.
Fig. 7.—X-ray of maxillary left side, July, 1932.

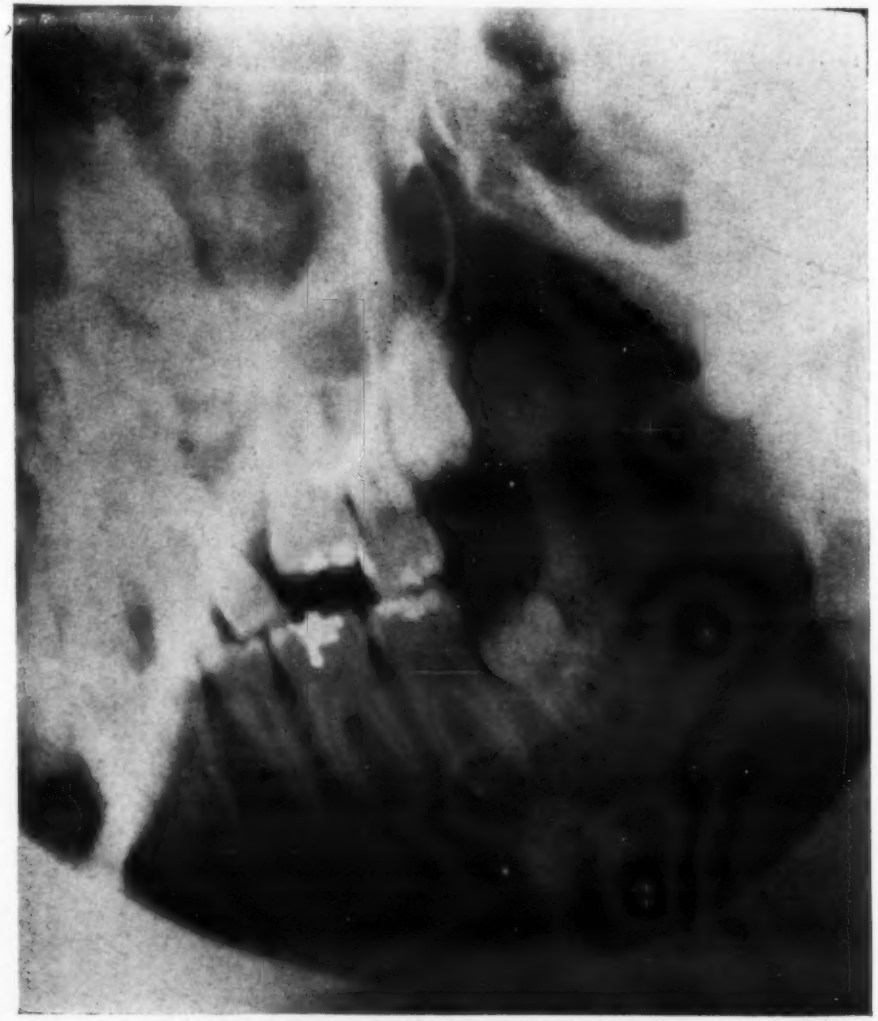


Fig. 8.—Extraoral view of left side, February, 1940.

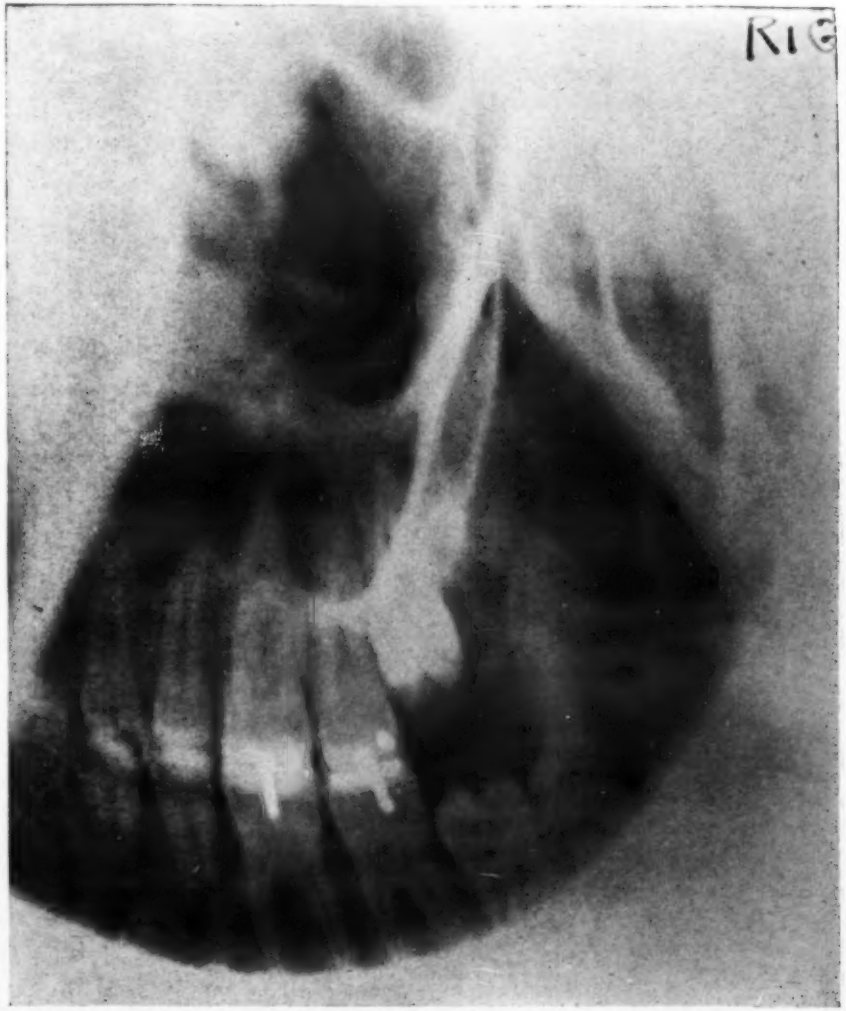


Fig. 9.—Extraoral view of right side, February, 1940.

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APICAL INFECTION VS. CYST

CHARLES B. HOPKINS, D.M.D., BOSTON, MASS.

IN THE past several years there has been an increased amount of research work in oral pathology, and many changes have taken place in regard to the technique of dental and oral surgery. Some conditions which had no special significance in the past are now regarded as surgical problems.

Many pathologic areas found through the use of x-ray have aroused a keen interest in the treatment of diseased conditions in the mouth. The experience gained by observation of pathologic areas about the apices of the teeth and bony structures proves that adequate treatment must be given in a very definite way if we are to be successful in cyst eradication; apical infection seems to be recognized as the etiological factor. Berger states that apical septic inflammations are usually dependent upon the decomposition of organic substances due to bacterial activities. Putrefaction may give rise to gases and acids which, passing through the apical foramen, cause irritation of the peridental structures. Broken-down roots are nearly always infected. They may act as physical irritants and cause inflammatory conditions in the alveolus.

According to Dr. Daniels, Dr. J. H. Henahan states that any tooth which has been nonvital for a long time, especially following root canal therapy, has caused pathological changes in the alveolar bone around it. There will be found a deposit of lime salts and a decrease in normal blood supply. This is the type of case in which we have a septic alveolus which on extraction results many times in an unfavorable postoperative condition and inflammation results.

A dental granuloma is the result of inflammation induced by the irritation of microorganisms or their toxins and is composed entirely of connective tissue with epithelial chains dipping down into the deeper strata which show tendencies toward cystic degeneration. The theory that they are the precursors of radicular cysts is maintained by some investigators and is suggested by clinical findings. Some method of systematized treatment must be used for their elimination. In spite of all the work done on teeth and mouth structures, surgical treatment given to diseases of the mouth does not equal that in other parts of the body. Observations from clinical work show the disastrous effects which result from apical infections.

Every tooth, if it is to be treated, should be watched carefully for the first sign of infection. This may help some cases that become dental problems. Three

important steps of treatment must be carried out. First, remove the cause. Second, secure direct drainage. Third, the focus of infection must be removed. There are three kinds of cysts. First, the radicular cyst is found associated with devitalized teeth. Second, the follicular cyst seems to develop around impacted or supernumerary teeth, or at times grows without any apparent association with dental structures. Third, the multilocular is a combination of cysts separated by a thin partition of bone, and it is the common theory that they have their origin in the enamel organ. They are found most generally in the mandible.

Patients who develop cysts report that most teeth, when extracted, were taken out because of either an abscess condition or infection. In checking these cases, I found that just taking a tooth out was not enough, as it does not remove the infected area. Only the cause is removed. In about 90 per cent of all cases, the cystic sac or granuloma is allowed to remain for nature to destroy. This is asking too much, and I firmly believe that all areas of infection found by x-ray at the apices of the teeth should be removed.

Blair says, "A blind alveolar abscess or one which has no route of discharge is limited to treatment and cysts are more likely to be formed in this than in any other class of cases. It is probable that a large proportion of these have more or less characteristic elements of cyst formation." Also, if the tissues about the apex of a root are once destroyed by suppuration, it seems quite certain that such a case never heals even though the cause is removed. The denuded cementum becomes the cause of the continuation of the disease.

There are too many teeth removed with little thought as to what may happen in the future. It is a well-known fact that almost all radicular cysts invariably are associated with infected teeth, and the earliest changes are in the peridental membrane, which soon leads to the formation of a granuloma. Infection then starts. Nature tries to wall off this activity and soon a cavity is formed, filled with connective tissue elements. Around the apex, there is bone destruction and root ends project into the radiolucent area. In the periphery, next to the bone, the soft tissue consists of fibroblasts and connective tissue fibers. The fibrous connective tissue is arranged in the form of a capsule around the pathologic area so as to wall off the latter from healthy tissue. The alveolar bone has been destroyed and occupied by fibrous tissue. As long as the inflammation is still active, it will spread and involve additional areas of bone and marked symptoms of osteoclastic activity take place on the bone lining the cavity. In about 50 per cent of all chronic inflammatory processes of the periapical region, epithelium is found in the granulation tissue. As stated by Kronfeld, in the majority of cases the epithelial rests of the peridental membrane are the source of the epithelium in dental granulomas and root cysts. Cysts develop from the epithelium in granulomas. It is impossible to draw a line between epithelium-containing granulomas and radicular cysts. In almost every case of granuloma with epithelium, one or several areas may be considered as the earliest stage of cyst formation. Therefore, it may be considered the earliest stage of potential cyst formation. Cysts can develop in two ways: an abscess cavity formed by inflammation due to breaking down of the tissues around the infected root apex, and through degeneration within the epithelial strands. The extraction of the tooth that caused the inflammation in the bone

by no means always causes the area to disappear. In fact, it may persist and such remaining areas may cause infection of the jaw. In other words, it cannot be expected that an area of infection which has developed over a period of years will always disappear after the irritating factor has been removed. The bone underneath the root end is different from the rest of the jaw. There are very small narrow spaces, and irritation from infected root tips must be held responsible. Therefore, it is probable that this area might remain after removal of the tooth. If this membrane is allowed to remain after extraction, the development of a cyst may continue, which forms the base for these sacs which merge with the pericemental membrane and bone trabeculae, and after extraction, unless removed either with the root or through surgical methods, many areas become walled off to continue to exist and grow. The removal of a tooth should never be entirely depended upon for the eradication of the pathologic areas. These areas seem to be more common in the maxilla. Why? Because there are 75 per cent or 80 per cent more teeth treated in the maxilla. The judgment of the operator and x-ray finding will, of course, be the basis for such treatment. Colyer states that the development of a cyst is slow but progressive and, as a rule, there are no disturbing symptoms unless it becomes infected or some structure, such as a nerve, is involved. Therefore, before the removal of any tooth, proper x-rays should be at hand. All granulomas must be held with suspicion and some means used to eradicate the diseased part. It is quite generally believed by all who have studied the subject of cyst development that such a lesion may be precursor of cyst areas. Therefore, according to Berger, it is of paramount importance that no part of the tooth or diseased portion of the peridental membrane which may act as a latent focus of infection should be left in the alveolus.

The operator who has this constantly in mind when studying x-rays of periapical lesions will tend to render a better service to his patient.

When cases are selected for treatment, a careful check at regular intervals should be made. When x-ray examinations of cases show infected or granulomatous areas at the apices of the roots, these should be removed at the time of extraction.

Boyd S. Gardner writes, "The mere extraction of infected teeth is not the only responsibility the operator has at the time such teeth are removed, as many patients fail to derive benefit unless the associated pathologic conditions are taken care of at the time the teeth are extracted."

When a tooth is removed, the diseased bone must receive the same consideration as infected areas elsewhere in the body. If not properly treated, the infection continues to spread as a rule, with no outward manifestation. Therefore, all teeth which have been treated for apical infection should receive x-ray examination yearly.

COMMENT

The bone is affected and, in many cases, destroyed by pathological changes due to inflammation at the root apex. Undoubtedly this is caused by retention of a granuloma, either after tooth removal or root canal therapy. During this period of destruction, there seem to be no radical symptoms to draw attention

to the lesion. Cysts, or rather cystic conditions, seem to be greatly on the increase. There must be a reason, so I advise that measures should be taken to eliminate all possible causes. The treatment of a cyst is thorough enucleation, and I believe most rarefied areas and granulomas should receive the same attention.

REPORT OF CASES

CASE 1, *Dentigerous Cyst.*—The patient was a man, aged 46 years. The tooth was treated by a dentist for a condition diagnosed as rarefied area at the apex of the root. Several months after treatment, the tooth became tender to mastication. The patient was referred for diagnosis and treatment.

X-ray.—X-ray examination showed the tooth slightly elongated and disclosed a large circumscribed area at the apex of the root (Fig. 1).

Diagnosis.—Cystic area which undoubtedly started from septic root apex.

CASE 2, *Granuloma.*—A man, 54 years of age, had had a tooth extracted two years previously by a dentist. The patient's face was swollen and first x-ray showed infection at the apex of the root. Several months later a slight swelling appeared on the gum. It was opened for drainage, but swelling recurred several times. The patient was referred for diagnosis and treatment.

X-ray.—X-ray examination showed a circumscribed area in the bone; on pressure exudate resembling pus was noticed (Fig. 2).

Fig. 1.

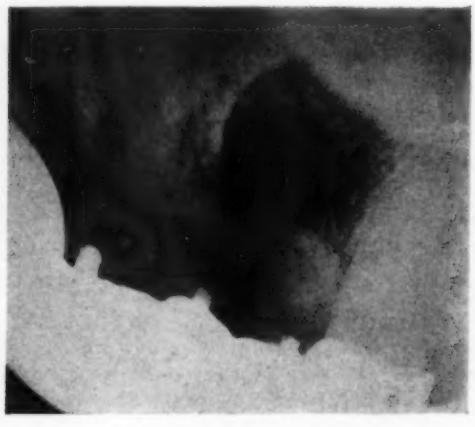

Fig. 2.

Diagnosis.—Infected cyst. If granuloma had been removed at time of extraction, this condition perhaps could have been prevented.

CASE 3, *Dentigerous Cyst.*—The patient was a woman, aged 22 years. A tooth was removed by a dentist about four years before. Slight swelling was noticed for six months in retromolar triangle and was treated by the dentist for local pericoronal infection.

X-ray.—X-ray examination disclosed that the root apex was undoubtedly fractured during extraction. The root had a circumscribed area and a sac was growing to wall off the infection (Fig. 3).

Diagnosis.—Cyst, due to irritation from infected root tip. All fractured roots should be removed.

CASE 4, *Granuloma.*—A woman, aged 30 years, had had a tooth filled by a dentist. It was extremely sensitive when the cavity was prepared. The filling was placed in the tooth without adequate protection of the pulp. The tooth remained tender to touch nine months afterward.

X-ray.—X-ray examination disclosed granuloma at the apex of the root (Fig. 4).

Operation.—The removal of the tooth, I believe, will not cure this condition. All the infected area must be thoroughly cleaned out to eliminate a possible recurrence or growth of a cyst.

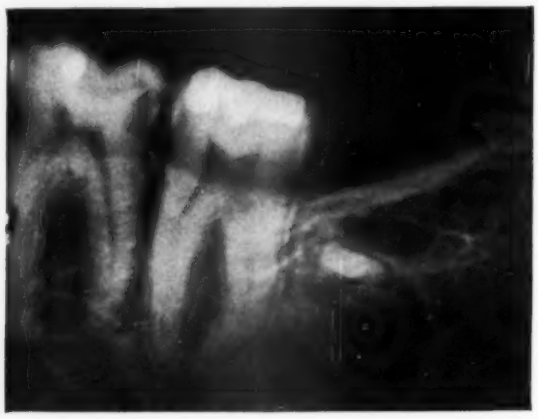

Fig. 3.

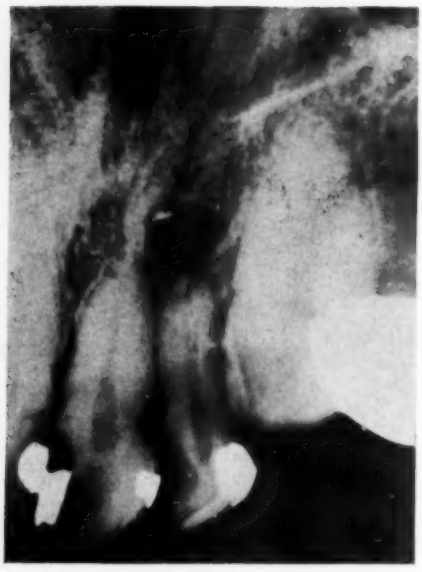

Fig. 4.

Fig. 5.

CASE 5, *Cyst.*—The patient was a woman 26 years of age. Several years ago a dentist had treated a maxillary right first premolar. The tooth was always tender after filling. The patient was told it would always be somewhat lame because of the severe abscess condition.

X-ray.—X-ray examination disclosed an infection at apex of the right first premolar and a large circumscribed area above all teeth from premolar to left central incisor (Fig. 5).

Operation.—If this tooth had been removed because of the severe infection and the infected area thoroughly treated, undoubtedly a healthy condition would have been created, thereby making unnecessary an extensive operation, with the loss of several teeth.

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CYSTS OF THE JAWS

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INTRODUCTION

CYSTS of the jaws have been classified etiologically, clinically, and histopathologically. No two textbooks agree on any specific classification. Pathologists find it difficult to differentiate microscopically between radicular and dentigerous cysts. Histologic diagnosis differs many times from the clinical diagnosis. Because of such differences, diagnosis and treatment can be aided by the adoption of a classification that is based on etiological facts and microscopic findings.

Basically, there are two types of cysts, those lined by epithelium and those lined by connective tissue.

I. CYSTS LINED BY EPITHELIUM

1. Radicular cysts
2. Dentigerous cysts
    (a) Follicular cysts
3. Multilocular cysts
4. Adamantinomas
5. Fissural cysts
    (a) Nasopalatine cysts
    (b) Median Maxillary cysts
    (c) Globulo-Maxillary cysts

II. CYSTS LINED BY CONNECTIVE TISSUE

1. Pyogenic cysts
2. Osteitis Fibrosa Cystica
3. Traumatic Bone cysts

1. Radicular Cyst

Synonyms—Dental cyst, root cyst.

Definition.—A radicular cyst is an epithelial lined cavity in a jaw bone containing fluid or semifluid contents and is characterized by arising from enclaved epithelial cells in the peridental membrane of a devitalized tooth root.

Etiology.—Theories as to origin have aroused much discussion. Gravitz[1] feels that the epithelial lining is derived from the oral mucous membrane. Each cyst starts with abscess formation within the granulation tissue (granuloma). Those supporting this theory believe that epithelization of the cavity cannot take place unless pus reaches the surface of the mucous membrane by means of a tract (fistula). The stratified squamous epithelium then grows inward and covers the cavity. The fistula may then close off.

Adloff and Schuster[2] favor the above, although Schuster believes that a proliferation of papillary downgrowth of oral mucous membrane may be cut off by invasion of connective tissue surrounding the downgrowth, and thereby becomes an "epithelial rest" in the periodontal membrane, and from this point invades a granuloma.

Malassez's[3] theory of epithelial rests is the most generally accepted. During the development and eruption of a tooth, remnants of Hertwig's epithelial sheath may become isolated in the peridental membranes (Fig. 1). Following the devitalization of a tooth, trauma or bacterial invasion may activate these epithelial rests or cells of Malassez. These activated cells form the cystic granuloma from which the radicular cyst develops.

Others feel that the presence of a solid granuloma is necessary at the apex of a devitalized tooth before a radicular cyst can be formed by proliferation of the epithelial rest.

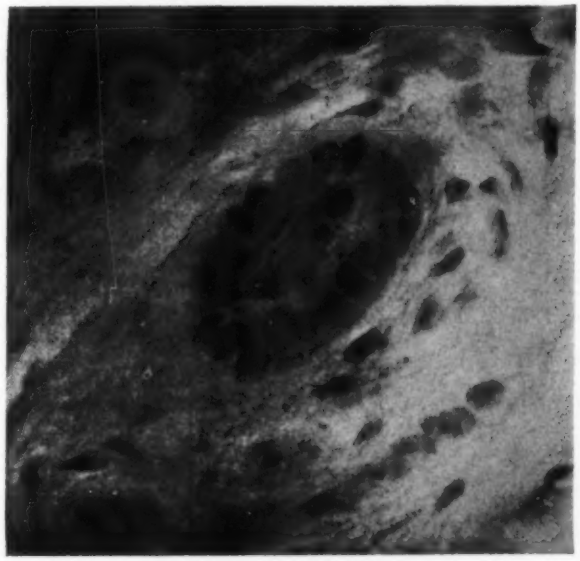

Fig. 1.—Epithelial rests surrounded by a delicate basement membrane. (Courtesy of Isaac Schour: From Dental Histology and Embryology, Noyes, Schour, and Noyes.)

Histopathology.—The radicular cyst is lined by a stratified squamous type of epithelium (Fig. 2). The body of the membrane is composed of fibrous connective tissue in which can be noted numbers of cellular elements dependent on the degree of inflammation. When the membrane is markedly vascular, cholesterin crystals are in evidence.

The cystic fluid varies from a light amber to a chocolate color. This fluid may or may not be sterile. When coagulation of the contents has taken place, it may vary from a semisolid consistency to that of cheese.

Long-standing radicular cysts may undergo secondary cholesteatomatous degeneration.

Symptoms.—There are no subjective symptoms until the cyst has reached a size able to exert pressure on the mandibular or other nerves of the jaws. It grows by expansion, resorbing the bone until the cortical, buccal, or lingual plates of the bone have been destroyed and the cystic sac has come into contact with the oral mucous membrane.

The radicular cyst may vary in size from a pea to a hen's egg (Fig. 3). When large, an external deformity of the jaw may be noted.

These cysts are subject to infection, and when acute, often present the symptoms of an alveolar abscess.

X-ray.—X-ray discloses a rarefied area, circumscribed by a well-defined white line called the lamina dura. It is usually associated with a devitalized tooth. When found in an edentulous area in which there is a history of a devitalized tooth or teeth having been extracted, it is known as a retained radicular cyst (Fig. 4).

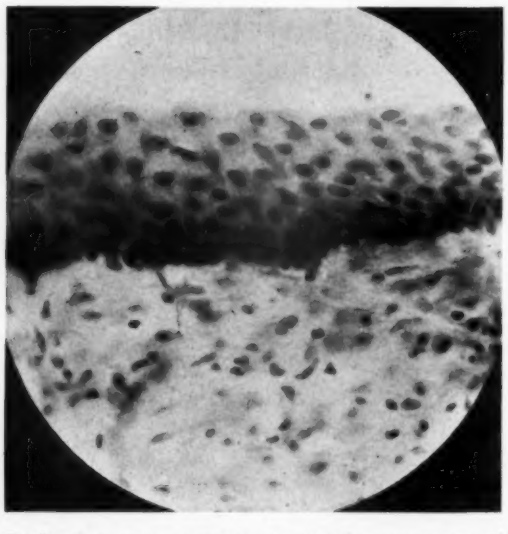

Fig. 2.—Radicular cyst showing stratified squamous epithelium.

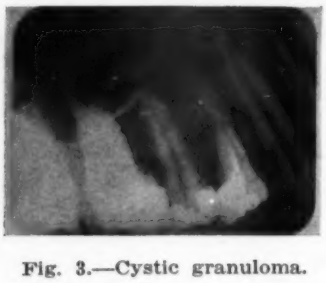

Fig. 3.—Cystic granuloma.

Treatment.—There are three methods for the eradication of a radicular cyst.

1. Extraction of the involved tooth with open operation for the enucleation of the cyst. If active infection is present, drainage must be established; otherwise the operated area is closed and organization of the blood clot permitted to take place.

2. The root canal of the involved tooth is treated and filled through the apex into the cystic cavity, and the cyst is enucleated periapically, with or without root amputation. This operation is usually limited to anterior teeth.

3. Partsch operation or one of its modifications following extraction of the involved tooth.

2. Dentigerous Cyst

Definition.—The dentigerous cyst is an epithelial-lined cavity containing fluid or semifluid contents and is associated with an unerupted or partially erupted, impacted, or partially impacted tooth.

Etiology.—Jacobs[4] has demonstrated the fact that many dental follicles or sacs contain epithelial inclusions (Fig. 5). Normally, the follicle disappears by atrophy following the complete eruption of a tooth.

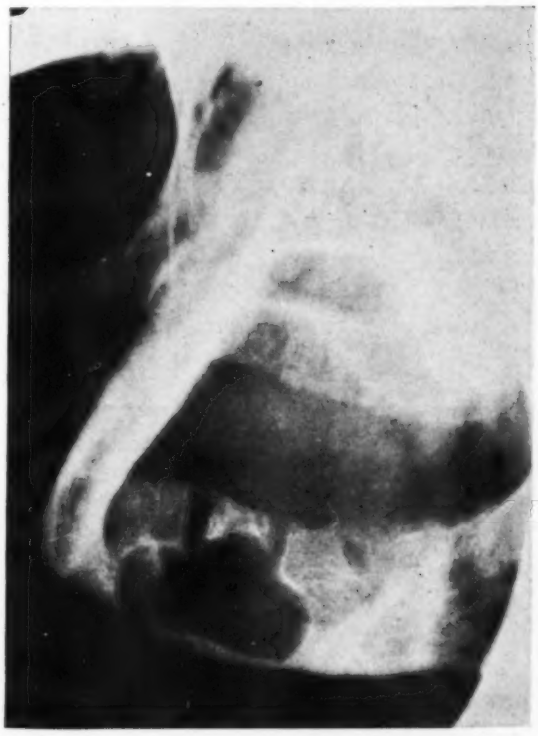

Fig. 4.—Retained radicular cyst of the mandible.

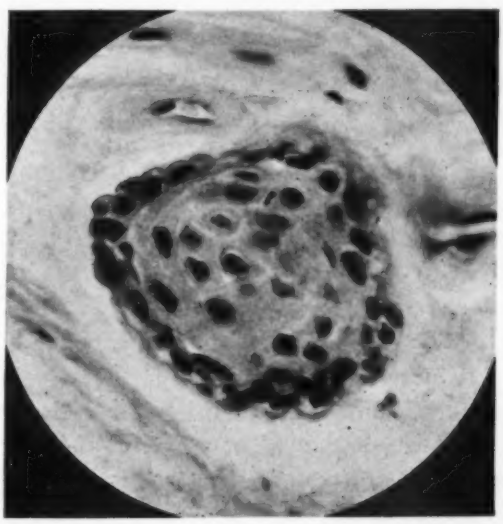

Fig. 5.—Epithelial inclusion in a dental follicle.

Incomplete disappearance of a follicle which contains epithelial inclusions may result in cyst formation, if proliferation occurs as a result of trauma or irritation.

Fischer[5] injected the follicles of deciduous teeth in cats with a silver nitrate solution and succeeded in producing a proliferation of the epithelial cells of the permanent tooth follicle, concluding that disturbances in deciduous teeth may activate disturbances in permanent teeth.

There is no evidence, however, that dentigerous cysts were produced by such procedures.

Histopathology.—The dentigerous cyst wall is composed of fibrous connective tissue lined with stratified squamous epithelium similar to that found lining the radicular cyst. Histologically, the dentigerous cyst cannot be differentiated from the radicular cyst (Fig. 6).

Diagnosis is based on the fact that dentigerous cysts are associated with the coronal structures of a tooth, and the radicular cysts are associated with the root elements of a tooth.

The cyst contents vary from fluid to semifluid. If the membrane is infected or very vascular, the color may range from yellow to bloody. Generally, the fluid is straw colored.

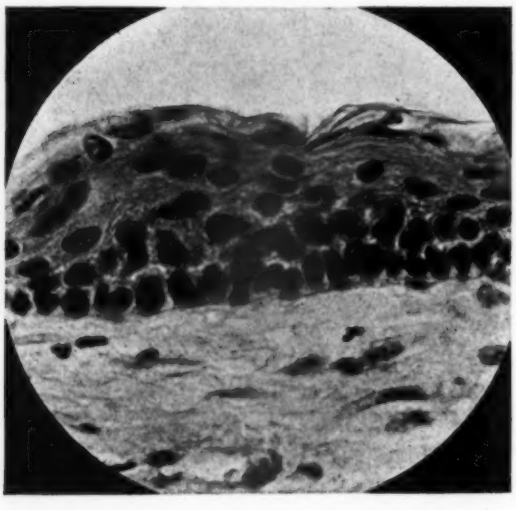

Fig. 6.—Epithelial cell membrane lining dentigerous cyst. Note the resemblance to epithelium of the radicular cyst shown in Fig. 2.

Symptoms.—Pain is present if the cyst has become infected, or if large enough to press on sensory nerves. The cyst occurs in those areas where unerupted or impacted teeth are most frequently found; namely, molar, cuspid, and incisor areas.

If large enough proportions are assumed, facial deformity may be the first symptom noted by the patient.

X-ray.—X-ray examination discloses a large rarefied area having its origin at the coronal surface of an impacted or unerupted tooth, circumscribed by a distinct line of demarcation unless infection is present (Fig. 7).

Treatment.—Treatment consists of enucleation of the cyst. If no functional relationship of the involved tooth can be attained if permitted to erupt, the tooth is also removed.

The Partsch operation should be resorted to, if too extensive surgery is necessary for eradication of the cyst.

2 (a). Follicular Cyst

Definition.—A follicular cyst is an epithelial lined cavity in the jaw bone filled with fluid or semifluid contents and is characterized by a history of an unerupted or partially erupted, impacted or partially impacted tooth having been removed.

Etiology.—Where an unerupted or partially erupted, impacted or partially impacted tooth has been extracted and the follicle retained in the jaw, proliferation of the follicle, if epithelial inclusions are present, may give rise to the formation of a follicular cyst.

Thoma[6] has suggested that an outgrowth from a dental follicle may become enclaved within the bone and proliferate into a simple follicular cyst. He has also suggested the formation of such a cyst arising from the follicle of a tooth which has failed to develop.

Histopathology.—The structure of the follicular cyst is identical to that of the dentigerous cyst.

Fig. 7.—Dentigerous cyst.

Symptoms.—The symptoms are those evidenced by the dentigerous cyst.

X-ray.—X-ray examination discloses the presence of a well-defined, circumscribed rarefied area revealing no association with any tooth (Fig. 8).

Treatment.—Treatment is the same as that of the dentigerous cyst, except that there is no involved tooth to be considered.

3. Multilocular Cyst

Synonym—Cystic adamantinoma

Definition.—The multilocular cyst is an epithelial-lined cavity filled with fluid or semifluid contents and is subdivided by one or more fibrous or bony septi, resulting in multiple locules.

Etiology.—Isolated islands of epithelium in the connective tissue stroma of dental follicles have been demonstrated, and it is conceivable that a tumor may arise from activation of these cells following some irritation.[4] (Fig. 5.)

The multilocular cyst occurs in those areas of the jaws where there exists an impacted, unerupted, partially impacted or partially erupted tooth, or where there is a history of such a tooth having been extracted.

Histopathology.—The true multilocular cystic membrane can be shown to be composed of small epithelial-lined cysts, epithelial pearls, giant cells, and xanthoma cells.

Symptoms.—The multilocular cyst may attain large proportions without causing pain. It is only when enlargement of the jaw begins to cause deformity that the patient may become aware of the abnormality. At times, it is discovered by routine dental x-ray examination. Pain occurs when pressure on nerve endings takes place or if infection intervenes.

X-ray.—X-ray examination discloses a rarefied area in which can be seen one or more narrow, sweeping septi subdividing the involved area into locules.

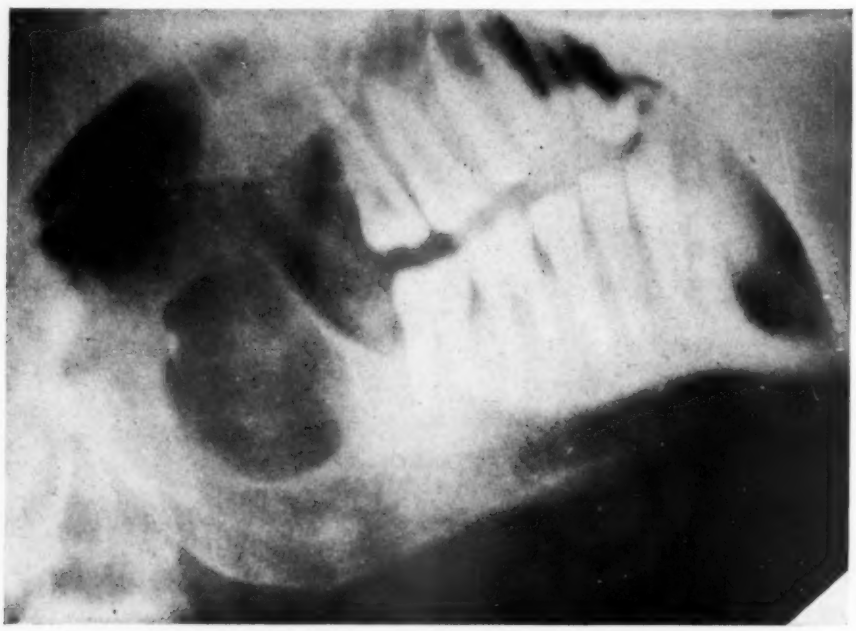

Fig. 8.—Development of follicular cyst following extraction of the third molar.

Treatment.—Treatment consists in the complete eradication of the cystic membrane. If this is not done, recurrence inevitably occurs. If there is any doubt, cauterization of the entire area is indicated. The patient must be followed up for a considerable length of time before being discharged.

Case 1.—The patient, J. S., was a male, aged 50 years. Five years before he was seen, he had had all of his teeth removed including a mandibular right impacted premolar. He had dentures constructed, which began to trouble him about two years later. Continual adjustments had to be made. Two weeks be-

fore admission to the hospital, his lower right jaw became markedly swollen, and he was treated for an acute infection by his physician, who performed intraoral incision and drainage.

Examination disclosed a marked enlargement of the lower right jaw. On palpation, a bulge extending from the cuspid to the third molar area could be felt. In places, the bone was parchment-like, and crepitus on pressure could be heard.

X-ray.—There was a large rarefied area in the right mandible extending from cuspid to about the third molar, and from the ridge to the border of the mandible. The entire area was marked by septi dividing the mass into numerous small cysts (Fig. 9).

Diagnosis.—Multilocular cyst.

Operation.—The entire mass was enucleated, the cavity cauterized and packed.

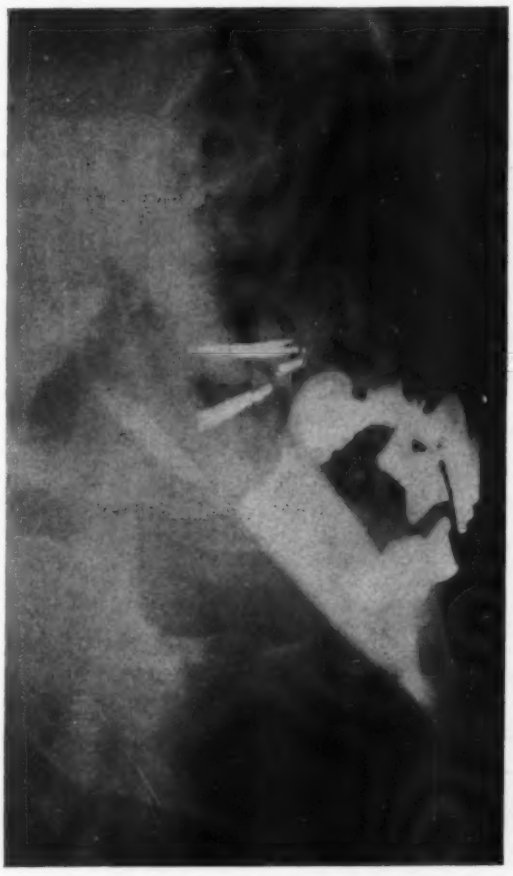

Fig. 9.—Development of a multilocular cyst following extraction of an impacted premolar.

Case 2.—The patient, M. Z., was a male, 40 years of age. Ten years previously he had had a left mandibular, partially erupted third molar removed because of recurrent infection of the tissues surrounding the tooth. For two years he had had vague, recurrent pain in the left side of the head and face. X-ray examination by his dentist revealed an abnormality in the left mandible.

The mandibular left third molar was missing. The second molar was loose, but vital as disclosed by vitality tests. The rest of the teeth in the left mandible were not remarkable.

X-ray.—There was a large radiolucent area extending from the vertical ramus to the first premolar, and from the ridge to the border of the mandible. The cortex on the lower border of the jaw in the third molar area was missing. In places the radiolucency was punctuated by darker areas. Thinned out septi surrounded these lesser regions (Fig. 10).

Diagnosis.—Multilocular cyst.

Operation.—Enucleation and cauterization.

Pathologic Report.—Microscopic examination of the cyst wall reveals it to be covered on one surface by a thin regular layer of stratified squamous epithelium. The superficial layers of this epithelium show considerable parakeratosis, and in places, there is a large amount of piled-up keratin. Beneath the epithelium, the connective tissue consists of densely arranged collagenous bundles, which are heavily infiltrated with lymphocytes and permeated by numbers of engorged capillaries. A few monocytes are seen. In the deeper surface of one of the cyst walls, there is a fibrin deposition. A number of nerves, pigmented macrophages, xanthoma cells, epithelial pearls, and giant cells lie in this fibrous-connective tissue (Figs. 11 and 12). The giant cells contain a large amount of pink homogeneous cytoplasm, with anywhere from twelve to twenty, small ringed dark nuclei in the central areas. There are cystic areas within the connective tissue, lined by a stratified squamous epithelium containing a granular pink-purple material with an admixture of nuclear debris in their lumina.

Diagnosis.—Multilocular cyst.

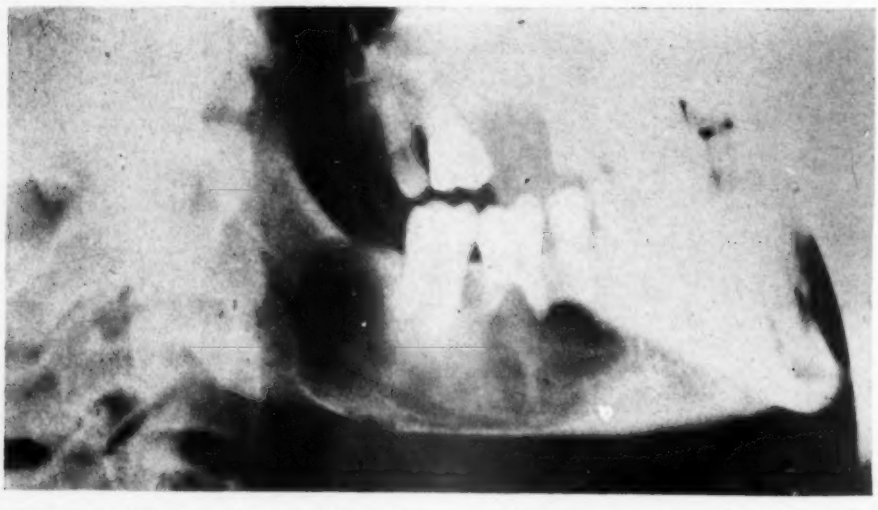

Fig. 10.—Development of a multilocular cyst following extraction of a third molar.

4. Adamantinoma

Synonym—Ameloblastoma

Definition.—The adamantinoma is a tumor of epithelial origin with locally malignant tendencies, characterized by the formation of small cysts lined by ameloblasts.

Etiology.—There are a number of theories endeavoring to explain the origin of these tumors. Malassez quoted by Padgett[7] believes that they arise as a result of a "proliferation of epithelial rests or epithelial debris."

Senn[8] feels that these tumors arise from the result of "embryonic inclusion of a matrix of epithelial cells."

Cahn[9] states that "the epithelial cells of the sacs of dentigerous cysts are similar to those of the inner enamel epithelium, and are adamantoblasts."

Scudder[10] believes that adamantinomas arise from epithelial rests or paradental debris.

Fig. 11.

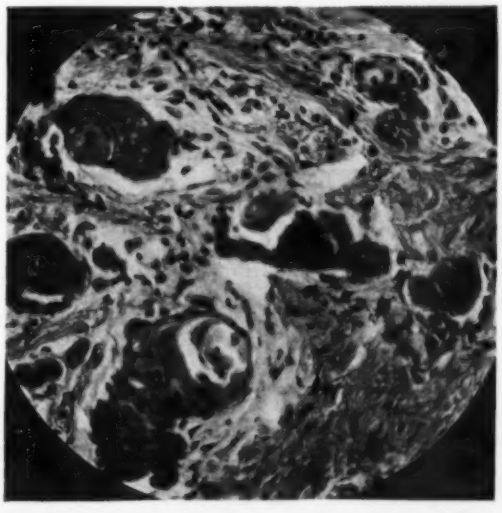

Fig. 12.

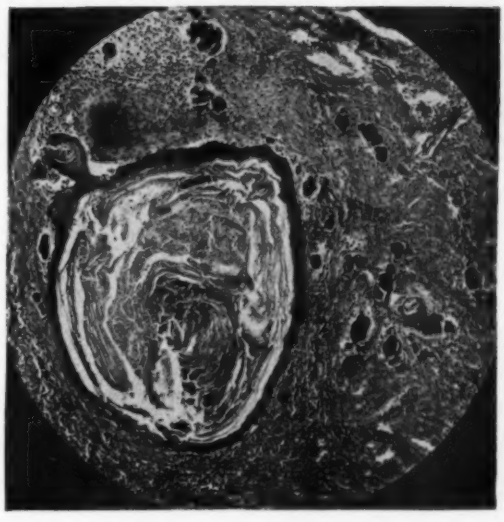

Fig. 11.—Xanthoma cells, giant cells, and epithelial pearls in a multilocular cyst wall. (High power view.)

Fig. 12.—Small cysts, epithelial pearls, and giant cells in the multilocular cyst walls. (Low power view.)

It is our opinion, following a study of a series of adamantinomas, that these tumors arise as the result of embryonic inclusion of epithelial islands in the connective tissue matrix of dental follicles or sacs, and they are proliferated to growth by some form of irritation.[4]

Histopathology.—Microscopically, the adamantinoma is composed of enameloblast lined cysts in the meshes of the stellate reticulum. Epithelial pearls are common and mitotic figures are occasionally found.

Symptoms.—There are no subjective symptoms, unless nerve involvement takes place. Attention is called because of deformity of the jaw caused by the expansive growth of the mass.

Treatment.—Complete eradication of the cyst wall and its contents is indicated. This is very important since these tumors are locally malignant and have a tendency to recur. Cauterization of the cyst cavity following enucleation is a valuable adjunct.

Case 3.—E. F., a female, was aged 29 years. Several years following the removal of a mandibular impacted left cuspid, the patient noted an increase in the size of her left jaw.

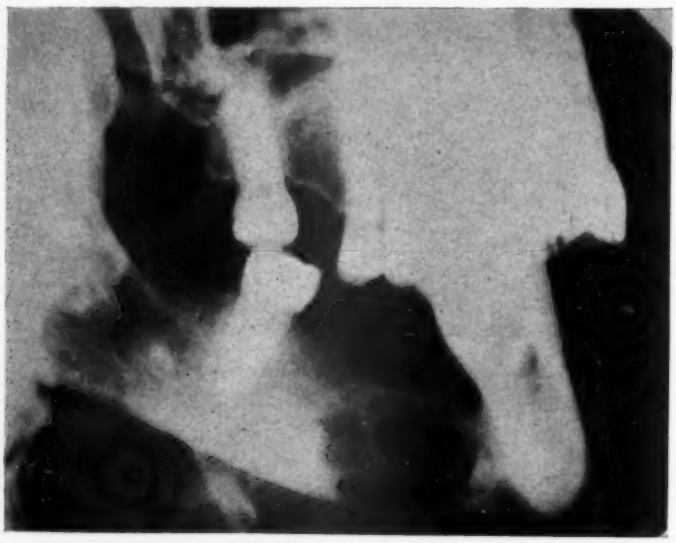

Fig. 13.—Adamantinoma following the removal of an impacted canine.

The left jaw was somewhat enlarged. On palpation of the buccal mandibular plate, a bulge could be felt extending from the cuspid to the first molar area. The buccal plate was parchment like and gave under pressure.

X-ray.—There is an irregularly defined radiolucency of the left mandible extending from about the cuspid to the first molar area. The radiolucency is marked by septi dividing the area into small cystlike regions. Both upper and lower borders of the mandible are involved. The septi are short and not well defined (Fig. 13).

Operation.—Enucleation and cauterization.

Pathologic Report.—Gross examination: Specimen is made up of a semicystic mass measuring 2 by 2.5 by 1 cm. The tissue is very pale. One section shows a grayish white, translucent, slightly mucinous cut surface.

Microscopic examination: Tumor is made up of irregular masses of elongated epithelial cells which, in the peripheral layer, are columnar in type. Some of the cell masses have cystic centers. In other groups are large cells which stain deeply with eosin and suggest squamous cells. Elsewhere definite cystic

spaces are present lined by deeply staining epithelium which varies from cuboidal to columnar. An occasional space is lined by a multi-layered epithelium resembling transitional epithelium. The stroma is made up of a moderately cellular connective tissue. Mitoses are few (Fig. 14).

Diagnosis.—Adamantinoma.

5. Fissural Cysts

(a) Nasopalatine Cysts

Synonym—Incisal canal cysts

Definition.—The nasopalatine or incisal canal cyst is a nonodontogenic epithelial lined tubular cavity filled with fluid when intact, and filling the incisal canal or patent nasopalatine canals when present.

Etiology.—Embryologically there are two sets of canals in the anterior maxilla; nasopalatine ducts and incisal canals.

The nasopalatine ducts generally disappear at birth. When they remain patent, there are two sources from which cysts may be formed.

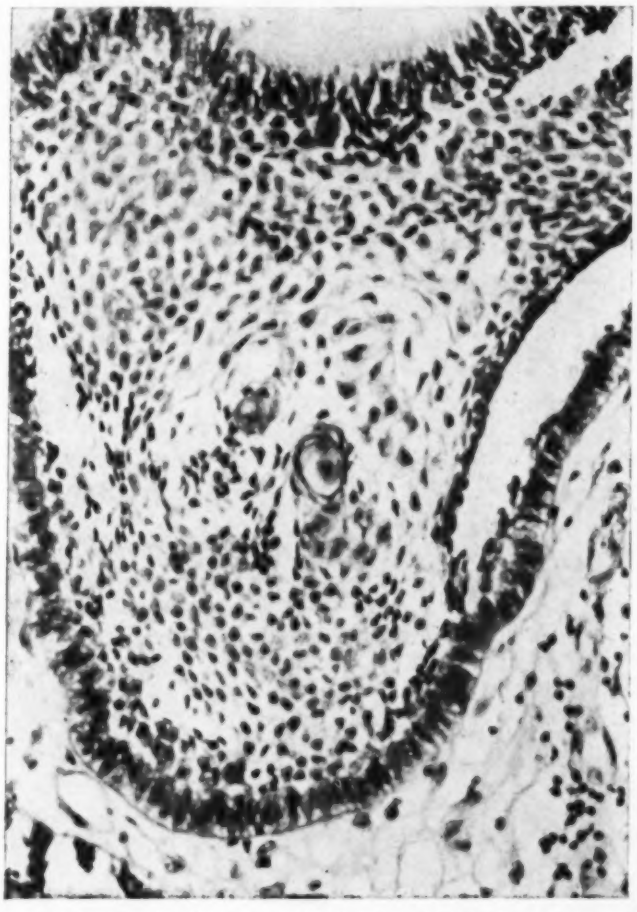

Fig. 14.—Adamantinoma. Cystic space lined by ameloblasts. Note epithelial pearl.

1. Epithelial inclusions may be left in the nasopalatine canal, which, on irritation later in life, may proliferate and form a cyst. The same is true of epithelial inclusions which may be left in the incisal canal.

2. Embryologically, before the nasopalatine ducts become shut off, there may be a herniation of the Schneiderian membrane of the floor of the nose. The superior portion of the ducts may become closed off, leaving remnants of the Schneiderian membrane within the duct which, on irritation, may proliferate and result in the formation of a cyst.

The same holds true for the incisal canal, except that the superior portion of the canal remains patent for the transmission of the nasopalatine vessels and nerves.

Fig. 15.

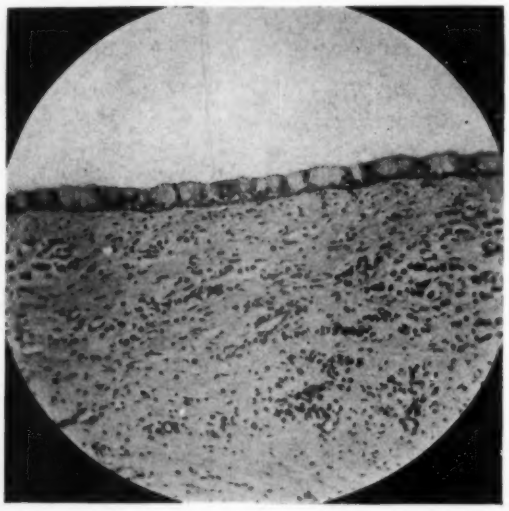

Fig. 16.

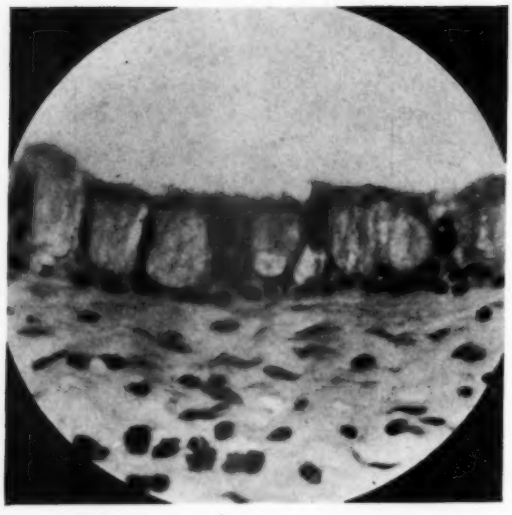

Fig. 15.—Columnar cells lining the incisal canal of the cyst. (Low power view.)
Fig. 16.—Columnar cells lining the incisal canal of the cyst. (High power view.)

Histopathology.—Because the epithelial inclusions or the herniation is derived from the Schneiderian membrane, the cystic epithelium is of the columnar variety and mucus glands can be demonstrated in the tissue (Figs. 15 and 16).

If the epithelial inclusions are those derived from the inferior tissues at the end of the nasopalatine ducts or the incisal canal, the cyst is lined by stratified

squamous epithelium. This accounts for the fact that in some of these cysts, columnar epithelium is found; while in others, only stratified squamous epithelium occurs.

Symptoms.—The nasopalatine duct cyst or incisal canal cyst may be symptomless for a long period of time and is found only on routine x-ray examination. When the cyst becomes large enough to fill the incisal foramen, the patient may notice a progressive enlargement of the tissue palatal to the central incisors.

If enlargement fails to occur, the patient may note a peculiar salty taste at times from this area, denoting drainage of the cystic contents through a minute fistula.

These cysts may become infected, resulting in swelling of the anterior palatal tissues, necessitating incision and drainage before removal of the cystic membrane.

Treatment.—When a nasopalatine or incisal canal cyst is discovered, surgical removal is indicated. There are two methods which may be used for its eradication: enucleation or marsupialization.

Enucleation is performed by retraction of the anterior palatal tissues exposing the bone in the region of the incisal foramen. A sufficient amount of bone is removed to permit visualization of the cyst, which is grasped with allys or tissue forceps, and the membrane separated from the bony canal walls and removed. If the cyst has been infected, drainage is indicated. If the cyst is aseptic, the palatal tissue may be returned to its place and sutured. Since these cysts are of nondental origin, extraction of teeth is not indicated.

(b) Median Maxillary Cysts

Definition.—The median maxillary cyst is a nonodontogenic epithelial-lined cyst filled with fluid or semifluid contents, arising in the median suture of the maxilla.

Etiology.—Before union of the palatal processes and the suture of the premaxilla takes place, there may be an inundation of the surface epithelium which later becomes closed off. Some of the epithelial cells become residual inclusion cells, and on irritation later in life may be stimulated to cyst formation.

Histopathology.—Because of its derivation from the oral epithelium, the median maxillary cyst is lined by stratified squamous epithelium. However, unlike radicular and dentigerous cysts, mucoid glands can often be demonstrated in the cystic membrane (Figs. 17 and 18).

Symptoms.—The median maxillary cyst may be present for some time without subjective symptoms. When infected, it is discovered because of pain and swelling palatal to the maxillary central incisors. Swelling may not occur when drainage takes place through minute fistulae. When small, the cyst may be mistaken for the incisal foramen. If aspiration with needle and syringe yields fluid, a cyst may be diagnosed.

X-ray.—X-ray discloses a well-defined, circumscribed, radiolucent area (Fig. 19); if it occurs in the anterior part, the roots of the incisors may be displaced laterally.

Treatment.—Eradication of the median maxillary cyst is either by enucleation or marsupialization.

(c) Globulo-Maxillary Cysts

Definition.—The globulo-maxillary cyst is a nonodontogenic epithelial-lined cavity filled with fluid or semifluid contents occurring generally between the maxillary lateral incisor and cuspid, but occasionally between the central and lateral incisors.

Fig. 17.

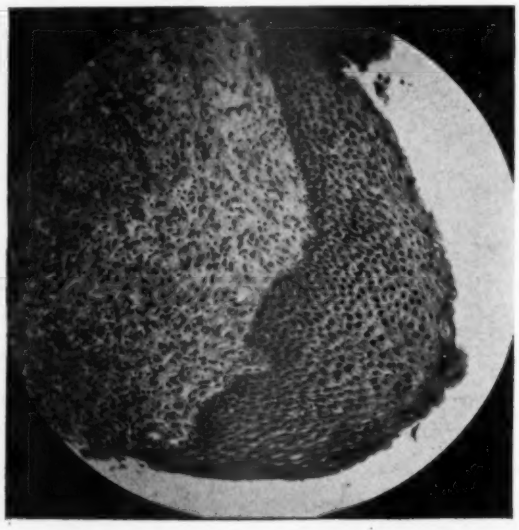

Fig. 18.

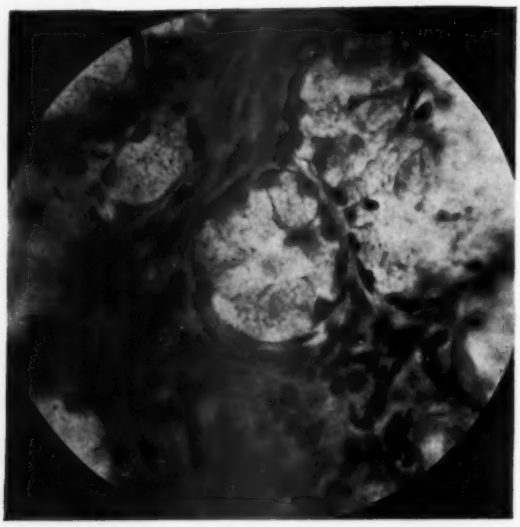

Fig. 17.—Median maxillary cyst with stratified squamous epithelial membrane.
Fig. 18.—Median maxillary cyst with mucous glands.

Etiology.—Embryologically, fusion takes place between the maxillary and globular processes. Untimely union may result in herniation of the Schneiderian membrane of the floor of the nose, this hernia later becoming closed off. Epithelial inclusions may enclave themselves in the fissure formed by these two processes, and later in life irritation may result in proliferation of the enclosed hernia or the epithelial inclusion, and result in the formation of a cyst.

Histopathology.—Since the epithelium is derived from the Schneiderian membrane, columnar epithelium and mucoid glands can be demonstrated in the membrane if the cyst develops from inclusions at the superior portion of the fissure.

Symptoms.—Progressively increasing size of the globulo-maxillary cyst generally causes a labial bulge in the region of the upper lateral and cuspid. No pain is complained of, unless infection occurs. Displacement of the lateral or cuspid, or both teeth, may take place.

X-ray.—A globulo-maxillary cyst in the presence of a devitalized tooth can be differentiated from a radicular cyst by careful x-ray interpretation of dental films taken from different angles. The questionable tooth can be displaced from the radiolucent area, and diagnosis established.

Diagnosis.—The presence of a cyst between lateral and cuspid, or lateral and central in the presence of vital teeth is significant of a globulo-maxillary cyst.

Treatment.—The globulo-maxillary cyst is eradicated by enucleation through the labio-alveolar plate, care being taken not to injure the teeth during operation.

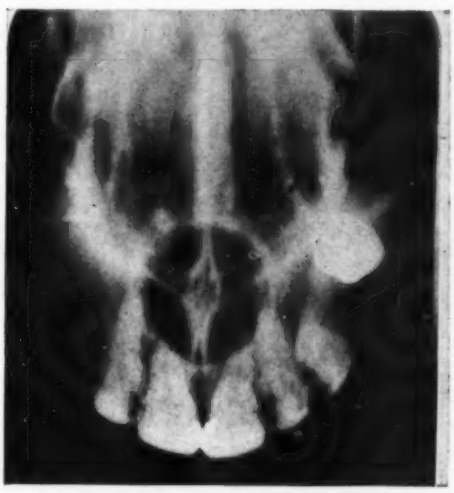

Fig. 19.—Median maxillary cyst.

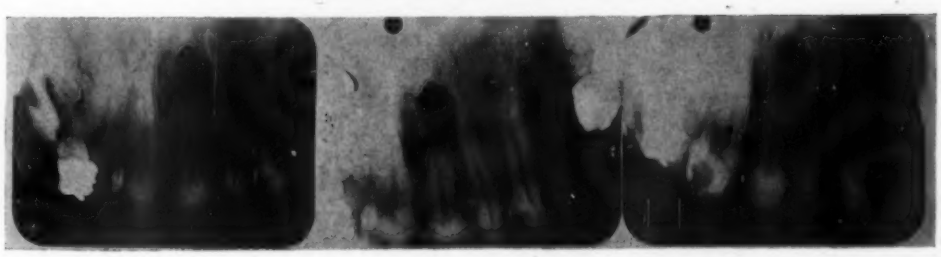

Fig. 20.—Globulo-maxillary cyst.

Case 4.—The patient, R. P., was a female, aged 40 years. The patient was referred for consultation because x-ray examination by her dentist disclosed a circumscribed, rarefied area over the apex of the left central incisor. No pain or other symptoms were complained of.

Clinical examination disclosed both centrals and left lateral incisor to have imperfect synthetic fillings. Careful electro-vitality testing of the anterior teeth revealed reactions within normal limits. The anterior teeth were all definitely vital.

X-ray films taken from different angles showed slight movement of the apex of the central away from the rarefied area. It appeared that this circumscribed area was posterior to the apex of the central and lateral to the incisal foramen.

In order to disprove cystic granuloma and prove this to be a nondental cyst, the left central was extracted and an open operation performed. No membrane or cavity was found in the apical area. It was only when considerable bone had been removed posteriorly and superiorly behind the apex of the socket that a cystic membrane was exposed and found to extend superiorly to the floor of the nose (Fig. 20).

II. CYSTS LINED BY CONNECTIVE TISSUE

1. Pyogenic Cysts

Synonym—Chronic walled off apical abscess, paradontal abscess, lateral abscess.

Definition.—A pyogenic cyst is a connective tissue lined cavity filled with pus, occurring on any part of the root of a tooth.

Etiology.—This process results from dental pulp infection when it occurs at the apex, and paradontal infection when it occurs on any one of the surfaces of a root.

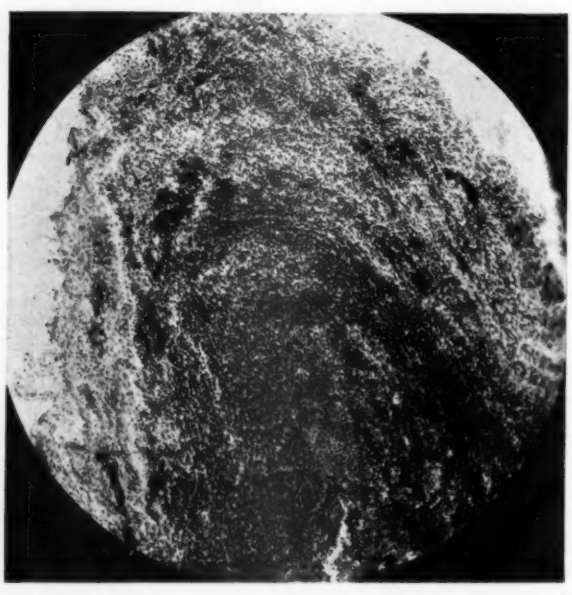

Fig. 21.—Pyogenic membrane showing marked round-cell infiltration.

Histopathology.—Nature's effort to localize an infective process is followed by the formation of a connective tissue membrane within which is an accumulation of pus. When the defensive factors of the body have abated the infection, fibroblasts arise from this connective tissue membrane and develop into osteo-

blasts which lay down osteoid tissue. After calcification, the involved area can be recognized in x-ray films as scar bone formation.

Microscopic examination discloses a connective tissue membrane, numerous small round cells and fibroblasts (Fig. 21).

Symptoms.—As long as the pyogenic cyst is under control, there are no subjective symptoms. Discovery is made after routine dental x-ray examination. When the process becomes acute, there is pain, swelling, temperature, and the other general symptoms of an acute alveolar abscess.

X-ray.—There is a circumscribed, radiolucent area not well defined and not marked by a lamina dura (Fig. 22).

Treatment.—It is unnecessary to remove a pyogenic cyst "in toto." Drainage of the area following extraction results in scar bone formation. If there is any doubt concerning the histology of the membrane, enucleation is indicated.

2. Osteitis Fibrosa Cystica

Synonym—Von Recklinghausen's Disease

Definition.—Osteitis fibrosa cystica is a disturbance of calcium and phosphorus metabolism and is characterized by hypercalcemia and hypophosphoremia, with resulting renal calculi and bone changes.

Etiology.—Because of hyperplasia or adenoma of the parathyroid glands, there is hypersecretion of the parathyroid hormone. As a result, calcium is withdrawn from the bones of the body into the blood stream, with consequent increased serum calcium and decreased serum phosphorus.

Normally in the blood stream there is 9 to 11 mg. per c.c. of calcium and from 2 to 5 mg. per c.c. of phosphorus. In osteitis fibrosa cystica, we find increases of the calcium level up to 16, and decreases in the phosphorus content down to 1 or less.

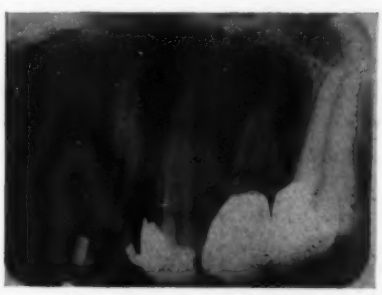

Fig. 22.—Pyogenic cyst from which the section in Fig. 21 was made. No epithelial cells in the membrane could be observed.

Histopathology.—From the dental standpoint, the depletion of calcium from the jaw bone discloses x-ray evidence of marked thinning. This is the first stage in the process and is interpreted as osteoporosis. If allowed to continue, cavities in the bones are formed which are found to be lined with connective tissue in contradistinction to most other jaw cysts, which are lined with epithelium, excepting pyogenic and traumatic bone cysts.

The bone is resorbed by osteoclasts which may form clumps of cells which present radiographic opacities and are called osteoclastomas.

Symptoms.—The patient usually presents himself to the physician with a complaint of pains in the bones and joints. The physician refers the patient to the dentist to rule out dental foci of infection. At times, attention is called to the disease only after mild trauma has caused a fracture of one of the long bones.

X-ray.—Routine dental x-ray examination for foci of infection may reveal multiple areas of osteoporosis, and in some places cystlike cavities, while in other places are more radiopaque areas of osteoclastomas (Fig. 23).

Treatment.—Blood calcium and phosphorus determinations in these cases will reveal disturbances in their levels, and this factor is pathognomonic of osteitis fibrosa cystica.

Removal of the hyperplastic parathyroid glands or the adenoma will result in recalcification of jaw bones without local intervention.

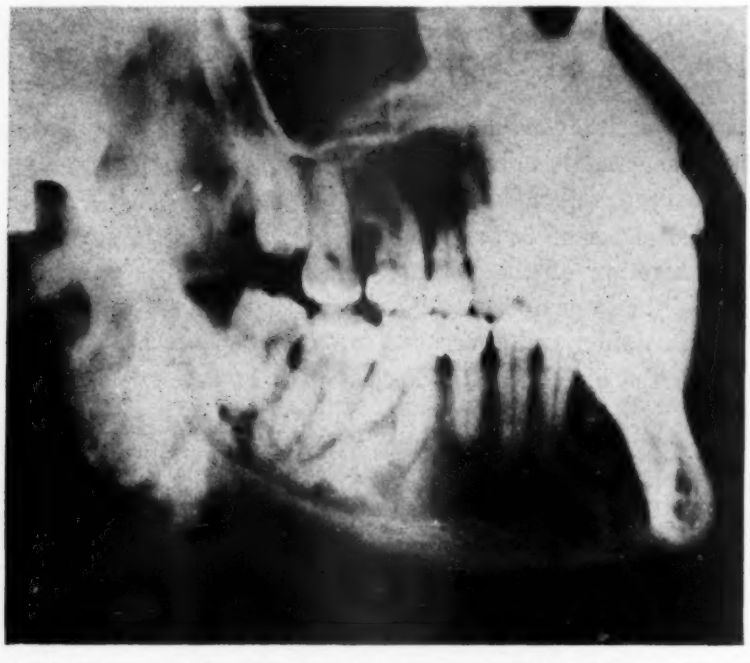

Fig. 23.—Osteitis fibrosa cystica showing osteoporosis, cyst formations, and osteoclastoma.

3. Traumatic Bone Cyst

Definition.—A traumatic bone cyst is a connective tissue lined cavity, the bottom of which is covered by the products of the decomposition of blood.

Etiology.—Blum, Ivy, and Thoma were among the first to recognize this entity. Trauma insufficient to cause fracture of the jaw but sufficient to cause intramedullary hemorrhage is the explanation given for its occurrence. Our cases have occurred following automobile accidents, and in a football player.

Histopathology.—The teeth in the area of the cyst are vital. When the cystic cavity is entered, there may or may not be an escape of bloody fluid. Only one of the cases we have operated on contained a bloody fluid. The other cases appeared to be hollow spaces in the mandible, on the floor of which could be seen shreds of reddish tissue resembling blood clot. The cavities were lined with a fine membrane not easily detachable from the bony walls.

Microscopic examination of the membranes disclosed connective tissue in the meshes of which could be found xanthoma cells.

Theoretically, the process in the formation of the traumatic bone cyst is one of progressive osteolysis. We have been unable to demonstrate osteoclasts in the bone spicules removed during operation.

It is possible that osteolytic enzymes released in the intramedullary spaces during and after the hemorrhage may be the means by which bone dissolution takes place, the connective tissue membrane being formed as a limiting agent of the process.

Symptoms.—For several days following injury to the jaw there is a complaint of soreness. There are no further subjective symptoms. The condition is generally discovered on routine x-ray examination of the teeth and jaws. There is no bulging of the lingual or buccal cortical plates. Osteolysis takes place in the anteroposterior direction.

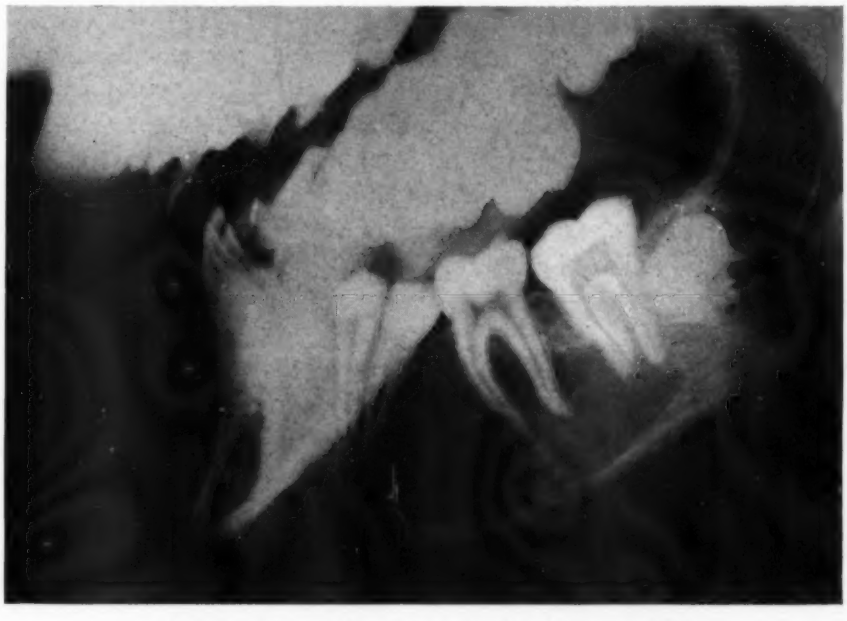

Fig. 24.—Traumatic bone cyst. All teeth are vital.

Diagnosis.—Diagnosis is simple if there are no devitalized teeth in the involved area. The history of a blow to the jaw, x-ray disclosure of a large rarefied area, all teeth in the area being vital, are significant of a traumatic bone cyst. In the presence of one or more devitalized teeth, diagnosis is difficult and can only be made during operation and microscopic examination of the contents of the cavity.

Treatment.—Treatment consists in opening through the cortical plate into the cavity and gently removing the floor contents. The connective tissue membrane need not be removed because fibroblasts springing from it take on the function of osteoblasts and lay down bone.

Case 5.—The patient, J. Z., was a male 16 years of age. The patient received a blow to the left jaw during a football game two years previously. He had always been in good health. During routine dental x-ray examination, a

large rarefied area was disclosed extending from the distal of the second molar to the distal of the second premolar, and from about the lower border of the mandible to the ridge. The area was punctuated by varying circumscribed densities and simulated the appearance of a multilocular cyst. Against this was the history of no tooth having been extracted in this area and no history of the presence of any supernumerary or impacted tooth. The third molar was unerupted and was beyond the involved area. All molars and premolars were vital (Fig. 24).

A curved incision with the concavity toward the ridge was made below the gingival margin buccally, and the mucoperiosteum reflected and retracted, exposing the cortical plate. This plate was removed below the apices of the molars and the opening extended upward between the roots of the second premolar and first molar. The bone was quite thin and was easily removed.

There was no fluid or contents of any kind filling the bone cavity. On the floor could be seen what looked like a blood clot which, when removed, exposed the mandibular vessels and nerve.

The bone cavity was lined by a glistening, delicate membrane which was difficult to remove intact.

Pathologic Report.—Microscopic examination revealed the membrane to consist of connective tissue, scattered throughout which were seen large cells with a small, central nucleus and light staining cytoplasm. The cytoplasm had a granular appearance, and in cases these granular appearances seemed to be droplets suggesting xanthoma cells (Fig. 25).

Other fragments of tissue were composed of blood clot. The bone showed nothing unusual.

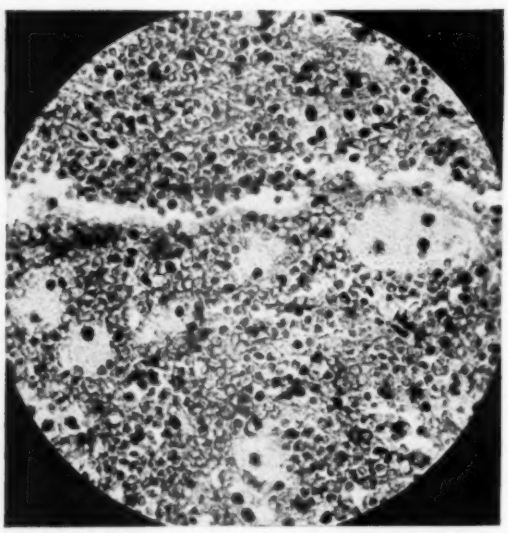

Fig. 25.—Traumatic bone cyst. Xanthoma cells and a blood clot can be seen in the meshes of the connective tissue.

CONCLUSION

An attempt to present a simple classification of cysts of the jaws has been made. A division into epithelial and connective tissue cysts with their subdivisions serves as an accessory means to diagnosis and an aid in determining a method of procedure for their removal.

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Case Reports

This month we have a case sent in by Dr. H. M. Brock which should be of especial interest to the dentist as well as the oral surgeon.

Case reports for this section of the Journal should be sent to Dr. Kurt H. Thoma, 53 Bay State Road, Boston, Massachusetts.

CASE REPORT NO. 40

THE EFFECT OF MAXILLARY IMPACTED THIRD AND FOURTH MOLARS ON THE CILIARY NERVES OF THE EYE

H. M. Brock, D.D.S., Port Angeles, Wash.

IN SUBMITTING the following case report it seems to me that both the dental and medical professions have been so intent upon the pursuit of infection during the past twenty years that they have not been conscious of the changes going on in the bony structure of the face and their consequences. Even Bing and Haymaker do not touch upon it in their late book on nervous diseases. In 1900 Dr. Black frequently used to find individuals who could bite three hundred pounds between a maxillary and mandibular molar on the gnathodynamometer, yet similar experiments performed a couple of years ago showed no such high rating. A more recent report shows a 13-year-old Eskimo girl to have a bite stronger than that of the best athletes in the University of Minnesota. This deterioration in both the bony and muscular tissues of the face is often bringing undue pressure on nerves, and producing effects remote from the seat of trouble.

A girl, aged 19 years, was referred to me by an optometrist. She was in a very nervous condition, having frontal headaches and double vision, with one image a little lower than the other. The x-rays which she had taken showed the mandibular third molars impacted and apparently pressing upon the mandibular nerve.

In the June, 1938 issue of the Journal of Orthodontics and Oral Surgery I gave a report on the effects produced upon the autonomic nervous system caused by pressure upon the mandibular branch of the fifth nerve, causing a reflex through the otic ganglion to the ninth and the vagus. So I will confine the present report to the maxillary division of the fifth. The x-ray showed not only both maxillary third molars impacted, but also impacted supernumerary fourth molars on each side.

Diagnosis.—The pressure upon the maxillary division of the fifth nerve was being reflected through the gasserian ganglion to the ophthalmic nerve, causing headache in the region of the supraorbital distribution. There was also a reflex

through the nasal branch of the ophthalmic to the ciliary nerves of the eye, causing the double and distorted vision.

Prognosis.—The removal of all of the impacted teeth would restore the patient to a normal condition in approximately four months.

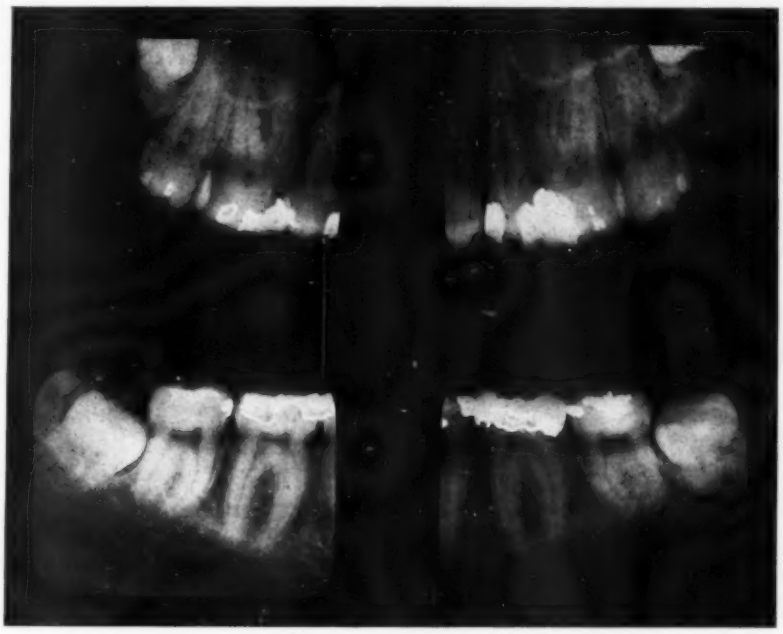

Fig. 1.

Treatment.—The teeth were removed by an oral surgeon with an almost immediate improvement in the nervous condition, and at the end of six months the eyes were perfectly normal.

Department of Orthodontic Abstracts and Reviews

Edited by

Dr. J. A. Salzmann, New York City

All communications concerning further information about abstracted material and the acceptance of articles or books for consideration in this department should be addressed to Dr. J. A. Salzmann, 654 Madison Avenue, New York City.

A Method of Measuring the Muscular Activity of Mastication: By Albert B. Larson, D.D.S., M.S.D., *Northwestern U. Bul.* April 1, pp. 20-22, 1940.

There have been several methods devised for the determination of bite-strength, but this does not indicate the true value of the amount of exercise obtained by chewing various foods.

It has been known for years that there is a small but definite local rise in temperature during muscular exercise. This is probably due to an increased blood supply, plus a certain amount of friction that naturally occurs. On this basis it was thought that if the temperature changes in the mouth during mastication could be accurately measured, these changes would be found to be a function of the exercise obtained by the mastication process. For this reason a thermocouple sensitive to .001° C. was prepared and the temperature changes in the mouth during mastication were measured.

It was found that during the first four to six minutes there was a rapid rise in temperature. The increase in temperature from then on was much slower, until within approximately six to twenty minutes when the maximum temperature was observed. It was found that the temperature increase was a direct function of the bite-strength. However, there was a definite gradation between the various foods used. The alternate contraction and relaxation procedure provided the least amount of exercise, while beefsteak provided the most.

Individuals with a strong bite-pressure in many cases raised the intraoral temperature as much as 1° C. Foods which require the maximum muscular contraction in order to be chewed thoroughly cause the greatest increase in temperature and the longest duration of increased temperature.

Practical Application. The gums and periodontal membrane are materially benefited by the increase in circulation through them. The amount of the benefit is dependent on the amount of exercise and this in turn on the strength of the bite-force and the toughness of the substance chewed. If patients are to profit by such exercises, not only must they be persuaded to give their chewing muscles and tooth-supporting structures adequate daily exercise, but also must dentists maintain the teeth (and supporting tissues) in good condition from the standpoint of chewing. Such practices should produce better developed

and better formed jaws in children and less periodontal disturbances and pyorrhea in adults.

E. H. H.

Effects of Natural and Refined Sugars on Oral Lactobacilli and Caries Among Primitive Eskimos: By Donald B. Waugh, D.D.S., and Leuman M. Waugh, D.D.S., With the Technical Assistance of Marjorie P. Waugh. *Am. J. Dis. Child.* **59**: 483, March, 1940.

A field study was made of the Eskimos of the lower Kuskokwim River area, in the southwestern region of Alaska. This region was chosen because of the opportunity afforded to observe both primitive and semicivilized natives.

SUMMARY AND CONCLUSIONS (BY THE AUTHORS)

The figures show the group fed various foods containing refined sugar to have been higher in dental caries at the beginning. This discrepancy may be accounted for in the dropping out of several Eskimos as well as in the fact that, in order to promote the necessary good will, the subjects were allowed to choose their own sweets. The foods were not divided by settlements but distributed as equally as possible among the people of all three villages. In many cases husbands and wives or other members of the same family selected different sweets. The percentage of those free from caries in the one group was compared with the percentage free from caries in the other, and the carious average of one group was compared with the carious average of the other. The results for the two groups are so drastically different that the deductions therefrom must be accepted as strongly indicative.

Practically 100 per cent of those free from caries in the group fed natural sugars remained free from caries, and showed a drop from the original percentage with lactobacilli. Of those with carious mouths in this group, 57.16 per cent showed an increase in cavities, with an average increase of 1.57 cavities, which might be considered a normal increase for actively carious mouths. From these figures one may conclude that with the Eskimo natural sugars do not initiate or cause an increase in the growth of oral lactobacilli; neither do natural sugars initiate or cause an increase in dental caries.

Of those free from dental caries in the group fed refined sugars, 72.73 per cent showed inception of caries, with an average of 3.6 cavities. Of those with carious mouths in the group fed refined sugars, 100 per cent showed an increase in cavities, with an average increase of 4.9 cavities, which must be regarded as an increase far above normal for a five- or six-week period. In the group fed refined sugar 81.90 per cent showed initiation of the growth of oral lactobacilli, and these organisms were present in 100 per cent of the mouths at the end of the feeding period. From these figures one may conclude that with the Eskimo refined sugars initiate and cause an increase in the growth of oral lactobacilli and that they also initiate and cause an increase in dental cavities.

This is the first recorded field work on the effects of natural and refined sugars on oral lactobacilli and dental caries among the Eskimos and must be regarded as preliminary.

Congenital Hypoplasia of the Mandible: By H. H. Weisengreen, D.D.S., and E. D. Sorsky, M.D. Fresno, Calif. *J. Pediatrics* **16**: 482, 1940.

A case of micrognathism which was responsible for cyanotic attacks in a newborn infant is described:

A female infant was delivered by cesarean section at the General Hospital of Fresno County. The parents were healthy, and their history was without significant data. The child was born at term and weighed 7 pounds. While it was apparent at once that the lower jaw was grossly retracted, the maxillae, dental ridges, and temporomandibular joints gave every evidence of being normal. No birth injury had occurred.

Shortly after birth, respiratory difficulties were noted, and examination showed the tongue to be resting almost against the posterior pharyngeal wall. All efforts to relieve the dyspnea by postural changes failed, and the choking fits increased in severity. This led us to transfix the tongue in the hope of giving temporary relief, even though we realized the general inadvisability of this procedure. However, despite strong supportive treatment including sedation and glucose, the infant died fifty-two hours after birth (Fig. 1).

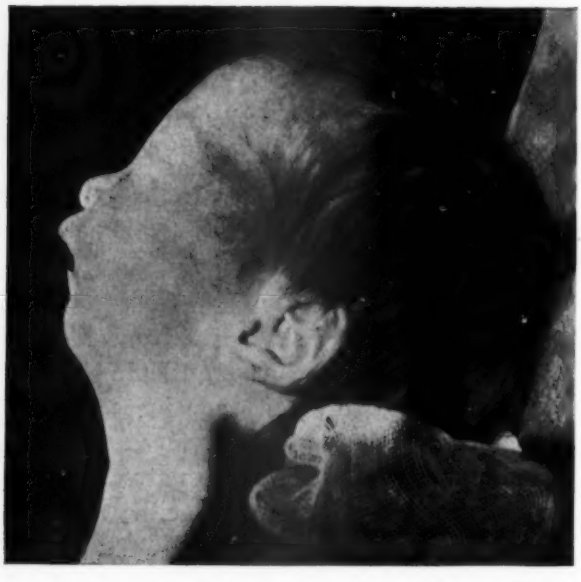

Fig. 1.—Case of congenital hypoplasia of the mandible.

Cases of congenital hypoplasia or agenesis of the lower jaw are a well-recognized deformity, characterized by extreme retraction of the mandible as a whole. The condition is noted at birth or soon after. The dwarfed development in the inferior maxilla causes a retracting chin while the upper jaw projects forward and slopes outward, thus emphasizing an abnormal relationship. The actual causal agency of this retardation of the lower jaw remains obscure.

From the twenty-fourth to the twenty-ninth days of fetal life the face is undergoing development. Projections grow out from the lateral boundary of the cephalic extremity toward a median line; these are called branchial arches. The superior arch, which with the frontonasal process is destined to form the

face, presents secondary protuberances that become the mandibular arch. These are followed by maxillary processes which form at the dorsal part of the mandibular arch and grow ventrally. Hence, anatomically, we may assume that this malformation is peculiar to the first branchial arch (Fig. 2).

Any situation which can influence adversely intrauterine life may prevent unification or coalescence of the branchial arches with resultant deformity. Gladstone and Wakeley have found collaboration for their belief in a pathologic origin created by deficient developmental vigor resulting from malnutrition of the fetus which may or may not include a toxic factor. Thus, from the standpoint of transmission, a disease state in the mother which causes a depressed growth impulse through defective nutrition is the inherited factor rather than the actual deformity.

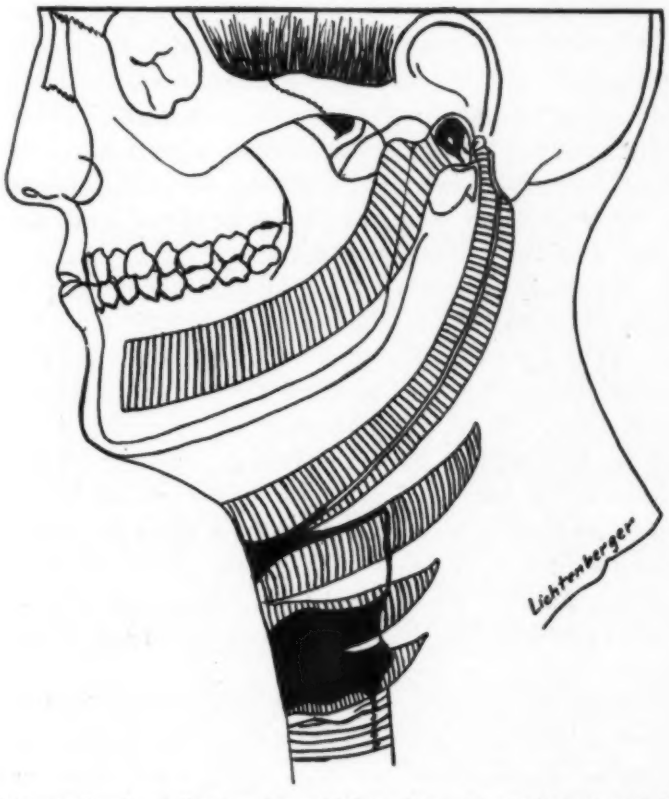

Fig. 2.—Metamorphosis of the five branchial arches. (According to Wiedersheim.)

On the other hand, several investigators, among them Davis and Dunn, express the opinion that these conditions may result from mechanical occurrences alone. This grouping would include not only the intrauterine stage, but the period of birth as well. In the uterus, pressure brought about by the increasing weight of the fetal body normally placed could easily affect the relative positions of the ununited maxillae and, at the same time, retard natural development. This explanation would seem to be strengthened in these cases by the frequent accompaniment of facial clefts which are known to be due to a failure of the maxillary processes to meet and unite. In this connection, too, it seems probable that abnormal positions in utero produce inhibitory

effects on growth. Still within this division may be added the traumatic injuries incidental to birth. Thoma has recorded instances of mandibular hypoplasia attributable to this cause.

In these conditions, the shortened lower jaw and accompanying inadequate musculature in the submaxillary region do not retain the tongue in a position sufficiently forward to permit normal breathing. Therefore, when the deformity is particularly marked, the tongue falls back against the posterior pharyngeal wall, and efforts of a temporary nature must be directed toward the maintenance of an air passage. Occasionally this is accomplished by keeping the infant in a position, usually face downward, that tends to overcome the effect of suction in breathing which pulls the tongue backward. When postural measures fail, however, more drastic means are attempted to avert the otherwise hopeless outcome.

Equally important is the necessity for establishing satisfactory feeding. The baby is frequently unable to grasp a nipple because of the uncontrolled tongue. Moreover, the opened mouth, constant salivation, and inability to swallow make attempts at artificial feeding dangerous. The situation is further jeopardized when a cleft in the lip or palate is present, a circumstance reported in the majority of cases. In addition, the enforced mouth breathing sends air into the bronchi unwarmed by passage through the nose, and a stimulus to respiratory affections is set in motion.

When the deformity is less severe, a partial air passage is maintained as well as indifferent success with feeding. Since, however, the baby usually gets only a portion of the nourishment required in this way, the physical condition becomes progressively worse, and medical aid is sought after malnutrition is in an advanced stage. Here, as in those cases requiring immediate therapy, every effort must be made to have the infant receive sufficient nourishment. The daily growth of the hard and soft tissues of the face will sometimes prove to be the only treatment needed. Lenstrup reported the case of a girl brought to the hospital at the age of 6 weeks. They were able to overcome feeding difficulties, and the growth following adequate nutrition so improved the musculature in and under the tongue that the organ remained forward in normal position.

The importance of the role played by the tongue in Robin's estimation may be surmised when, in a more recent article, he quoted himself as having first used the term "glossoptosis" in 1923. Both he and Lapage emphasize the influence of the nursing mother's posture, and use as an illustration the fact that the young of certain mammals reach up to nurse. In explaining what he called orthostatic feeding, Robin says, "The baby sucks with his thorax held up straight so that he lifts his neck and propels his chin forward. . . . In order to feed her child orthostatically the mother is obliged to hold herself erect, with the breast elevated and the thorax held up." Otherwise, the weight of the breast has a tendency to rest on the child's chin. This idea of feeding an infant in a manner that will enforce action in the muscles of the lower part of the face has been applied also by Davis and Dunn, who attached to a nursing bottle a curved plate that rested against the infant's upper lip. This plate, being freely movable, could be regulated to make the child extend the lower jaw.

Types of apparatus much more complicated than this have been devised and have found favor with some physicians. Eley and Farber constructed a brace that "consists of a strong piece of copper wire which rests on a soft flannel band as it passes across the forehead and which is shoed with rubber tubing as it passes down the side of the jaw. Here it passes under the ramus, forming a right angle to the descending limb. The two ends of the frame are then joined by a piece of white tape which holds the frame firmly in position and tends to maintain the pressure that is exerted against the mandible and the floor of the mouth. The mandible is then displaced forward and, at the same time, pressure is exerted against the floor of the mouth and the base of the tongue." They claim satisfactory results with this apparatus.

Callister is under the impression that his application of skeletal traction to the correction of micrognathy in an infant is unique. An apparatus resembling the jury-mast used for fractured cervical vertebrae was strapped to the child. From this a large flat aluminum ring was brought around in front of the baby's mouth. Arranged with a spiral spring to allow some movement, "a thick silver wire was passed through the skin and around the mandible" and fixed to the metal band. According to the author, the child wore this for four weeks, after which it was no longer needed; she was discharged in excellent condition.

CONCLUSION

Cases of congenital hypoplasia, it appears, require either emergency treatment from the time of birth to maintain respiratory adequacy or, when the deformity is less severe, therapeutic intervention after a period of days or weeks in cases where insufficient nutrition has inhibited normal development.

Measures designed to correct the deformity by nonoperative methods as soon as the baby's physical status permits have been found preferable to surgical or mechanical procedures carried out later in life.

While various forms of intricate apparatus have been devised and have been reported upon favorably, they have failed in many instances, owing to the restricted area of application, the tenderness of the tissues involved, and the danger of increasing respiratory obstruction.

With the exception of emergency measures, treatment, it would seem, is most successful when directed toward the establishment of a natural bodily development in the child in order to encourage growth in the underdeveloped region.

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Editorial

Report of the Chicago Meeting of the American Association of Orthodontists, 1940

THURSDAY afternoon, May 16, 1940, the "Limited Attendance Clinics" of the American Association of Orthodontists drew the curtain on the Thirty-Eighth Annual Meeting of the Association. The meeting, held at the Edgewater Beach Hotel in Chicago, opened with the various committee meetings on Sunday, May 12, and lasted through Thursday, May 16, before it was decided that the time had arrived for the thirty-eighth consecutive meeting of the organization to close for another year.

Chicago and the Edgewater Beach Hotel are by no means new to the American Association of Orthodontists. In fact, this organization has held at least seven of its meetings in Chicago and at this same hotel in years gone by. It is recalled, meetings were held at the Edgewater Beach when the membership and attendance were not one-fifth of what they were at the 1940 meeting; therefore, the Edgewater at Chicago is somewhat traditional as a meeting place for the American Association of Orthodontists.

The activities got away to a good start on Monday morning with the annual recreation and get-together golf tournament held at the Tam O'Shanter Golf Club. On the same day the annual American Association of Orthodontists trap shoot was held, and in the evening the membership enjoyed the annual golf stag banquet. If the attendance at this event was a criterion, the excellent entertainment offered by the local golf committee during the gala evening that ensued was indeed a great success.

The program presented in Chicago has been outlined previously in the May issue of the AMERICAN JOURNAL OF ORTHODONTICS AND ORAL SURGERY. The meeting was interesting; the program, timed with dispatch and precision, was unusually well attended and received. The innovation introduced at this meeting, the holding of the clinics in the morning on the opening day, seemed to strike a highly receptive note with the membership at large, because it provided ample time without haste and confusion for the members to absorb and digest many of the fine clinics which had been prepared far in advance of the meeting date. It was thought by many that the plan reflected the added advantage (as compared with the old custom) of not being a counterattraction and in conflict with the rush for trains by the members on the last day of the meeting. Much comment was heard that this arrangement is a step forward in the general plan of the meeting of the Association and that it should be followed out in the future, because many members feel that the clinics of this society are one of the very high spots of the meeting and are always well worth attending religiously with notebook in hand.

On Tuesday afternoon, Dr. Franklin B. Snyder, President of Northwestern University, made the address of welcome, and this was responded to

by President-elect, Henry U. Barber of New York City. President Wm. A. Murray then delivered the President's address, and the remainder of the afternoon was devoted to the regular scientific program of the day.

Wednesday morning was devoted to the scientific program, and this was followed at 12 o'clock noon by the past-president's luncheon. After listening to the Wednesday afternoon contributions to the scientific sessions, most of the members attended the President's reception, banquet, and floor show in the evening, which later was the climax of the entertainment features of the meeting.

The remainder of the session was devoted entirely to the scientific program. At the last executive session of the Association which was held at 4 P.M. Thursday afternoon, it was voted to hold the 1941 annual meeting of the Association in New York City (date to be announced later). The officers chosen to lead the Association for the coming year are as follows:

President—Henry U. Barber, New York, N. Y.
President-Elect—Dr. Claude R. Wood, Knoxville, Tenn.
Vice-President—Dr. G. W. Grieve, Toronto, Can.
Secretary-Treasurer—Dr. Max E. Ernst, St. Paul, Minn.

Dr. James D. McCoy of Los Angeles, Calif., was elected to the American Board of Orthodontics for a term of seven years.

The American Association of Orthodontists, long regarded as one of the most vigorous, fraternal, and progressive societies in dentistry, closed one of its usual enthusiastic meetings. The meeting reflected loyal and painstaking preparations on the part of all officers and committees, and, if the advance enthusiasm of the New York contingent is an indication, the members of the American Association of Orthodontists may again look forward to another outstanding meeting in New York City in 1941, with probably the largest attendance in its history.

H. C. P.

News and Notes

George B. Winter Memorial Issue of the Archives of Clinical and Oral Pathology (September, 1940)

The Council of the Archives has decided to dedicate the September issue to the memory of Doctor George Ben Wade Winter of St. Louis.

This number is to contain articles contributed by his friends and students on the subject of the impacted third molar. These will deal with the clinical, embryological, anatomical, pathological and roentgenological viewpoints, as well as editorial and historical contributions.

The following have consented to contribute to the issue:

Dr. Arthur M. Alden
St. Louis, Mo.
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Richmond, Va.
Dr. J. Holden Beckwith
Miami, Fla.
Dr. Theodor Blum
New York, N. Y.
Dr. Victor Boyko
Paterson, N. J.
Dr. Elmer H. Brown
Trenton, N. J.
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Brooklyn, N. Y.
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Philadelphia, Pa.
Dr. George W. Christiansen
Detroit, Mich.
Dr. Thomas J. Cook
Philadelphia, Pa.
Dr. Wells A. Daniels
Springfield, Mass.
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Dr. Kurt H. Thoma
Boston, Mass.
Dr. Earle H. Thomas
Chicago, Ill.
Dr. A. P. Williams
Louisville, Ky.

The New York Institute of Clinical Oral Pathology

Tenth Meeting of the Biological Photographic Association

The tenth annual convention of the Biological Photographic Association will be held at the Hotel Schroeder, Milwaukee, Wis., Sept. 12, 13, and 14. This society is interested in the further study of photography as applied to the biologic sciences, and the improvement of its technique. Scientific photographers from all parts of the country will meet to exchange ideas and information on still and motion picture photography as well as the latest developments in color work. Formal papers will be presented outlining new methods of technique, and there will be informal round table discussions which will be especially instructive. Commercial firms specializing in the manufacture of scientific photographic apparatus and materials will exhibit and explain the use of their products. A salon consisting of natural color and monochrome prints of biologic and clinical subjects will illustrate a very fine degree of perfection in biologic photography.

Attractions at A. D. A. Meeting

The eighty-second annual session of the American Dental Association to be held in Cleveland, September 9 to 13, will mark the centennial of American dentistry. Special emphasis is being placed on the Hall of Exhibits, so that it will truly present a comprehensive and graphic account of the contributions made by the profession in ten decades.

More than 125 separate exhibits are already being prepared for display in the new $12,000,000 Cleveland Public Auditorium which will house the convention's sessions, clinics, lectures, and demonstrations as well as the Hall of Exhibits. Divided into three sections—science, health, and history—the displays, together with technical exhibits, will encompass the whole world of dentistry.

The American Dental Association Historical Exhibit will portray the development of national dental organizations in America. Beginning with the formation of the first American dental society, 100 years ago, the exhibit will present all of the evolutionary changes that have taken place and resulted in the American Dental Association as it is in 1940.

The principal events and dates in national dental organization history will be depicted by large plaques, and copies of the original constitutions and by-laws of the various national dental societies will be displayed together with a rare collection of photographs of past presidents of the A.D.A. from 1859 to the present.

Visitors to the Hall of Exhibits in Cleveland will be able to review the past as far back as Egyptian days, at displays of ancient dental instruments. The various stages of dentistry's development will be portrayed through the years down to the present time, which will be represented by exhibits of the most advanced results in research and practice.

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*The Journal will make changes or additions to the above list when notified by the secretary-treasurer of the various societies. In the event societies desire more complete publication of the names of officers, this will be done upon receipt of the names from the secretary-treasurer.

†The Journal will publish the names of the president and secretary-treasurer of foreign orthodontic societies if the information is sent direct to the editor, 8022 Forsythe, St. Louis, Mo., U. S. A.